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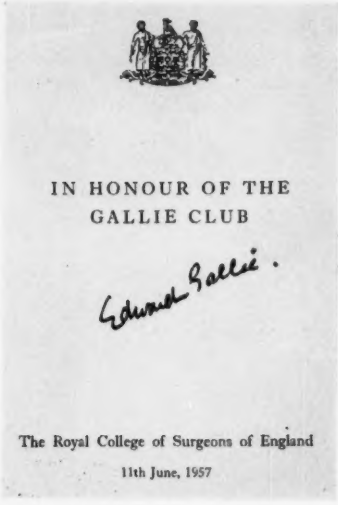
EDWARD GALLIE

SEVENTY-FIFTH BIRTHDAY

Edward Gallie recently celebrated his seventy-fifth birthday in London. Despite the vast influence that he has exerted for so many years in Canada and the United States, London is perhaps still his spiritual home. He was accompanied by members of the "Gallie Club" and many friends in this country to whom he has been so kind, and who so often have been proud to pay homage to him.

EDWARD GALLIE

Edward Gallie is a general surgeon; he always was a general surgeon and still is. But I still remember very clearly a luncheon in Toronto when he goaded and taunted a neurosurgeon, plastic surgeon, rectal surgeon, urogenital surgeon, upper-abdominal surgeon, orthopaedic surgeon and others to debate the merits or demerits of specialisation and generalisation. He had his tongue in his cheek; he knew that although priding himself as being a so-called "general surgeon" his first love was to that part of general surgery known as orthopaedics. Nevertheless his disciples and students now working throughout the length and breadth of Canada have the training by which, at any time, generalisation can turn into specialisation.



With great loyalty, Edward Gallie chose London to celebrate his seventy-fifth birthday where he was accompanied by nearly every member of the Gallie Club who are all his "galley-slaves." It was in London that he himself slaved hard to achieve the Fellowship of the Royal College of Surgeons of England; and his students, associates and friends from both continents joined him in London, Oxford and Edinburgh in happy social activity as well as in occasional academic pursuit. His dinner birthday party was in the oak-panelled hall of the Council Room of the Royal College of Surgeons of England, the oak tree having been planted and grown in the reign of Queen Elizabeth I, and the panelling from it dedicated after all these years by our monarch, Queen Elizabeth II. It was in this College that Edward Gallie received its very greatest award and honour—the Gold Medal—awarded less than forty times in a century. We all thought that he went home to Toronto with much of the glow and warm happiness that we had enjoyed by being in his company.

EDITOR.

THE TREATMENT OF PERTROCHANTERIC FRACTURES

The special feature of pertrochanteric fractures is that it is mostly a problem of old age, and that the comfort of the patient, the ease of nursing and the diminution of the complications of decubitus make desirable the earliest possible freedom of movement, if not ambulation. Compared with transcervical fractures pertrochanteric fractures rarely lead to avascular necrosis, but in the latter the mechanical difficulties of shearing force are found in a much more acute form. This has not always been sufficiently recognised. In a reduced transcervical fracture held by an intramedullary nail the muscular forces applied to the trochanters have an impacting rather than a disrupting influence; but in pertrochanteric fractures the relationship is altered in favour of disruption. In the transcervical fractures the intramedullary nail is mechanically well able to deal with the forces applied; but, because a pertrochanteric fracture occurs at the end of the cervical lever and therefore at the meeting point of the extremity of shearing force with the focal point of muscular power, the position can be held only by a device that must be angular to conform with the anatomical structure and must be capable of resisting the combined forces of the weight of the attached limb (rather less than a quarter of the total body weight) and the internal motive forces of muscular activity. It is an intriguing mechanical problem. Nail or screw devices passing through the skin and attached to an external framework, while having some mechanical advantage over simple external traction methods, did very little to solve the problems of decubitus and the needs for early ambulation. The original idea of the angulated appliance or nail-plate fixed

directly upon and through the site of fracture appears to have come from Neufeld. In its original form it comprised a relatively thin piece of steel, the lower half of which was concavo-convex in section to fit the outer side of the femoral shaft and the upper half was bent at an angle and was forged into a cross-sectional V-shape. There were several early modifications, such as those of Jewett, Moore, Blount, and Capener. The last, which is commonly used in Great Britain, was designed to provide greater strength at the angular junction. Unfortunately at one time some manufacturers found it easier to make this device in two pieces welded; and so the nail-plate exhibited all too clearly the mechanical weakness engendered by the internal body forces above mentioned, and emphasised that V-nail plates must be forged out of one solid piece of metal reinforced at the angle. The accurate insertion of these one-piece angulated devices is at first not easy to the unpractised surgeon, although it is not at all difficult if a few straightforward precautions are observed. To meet this supposed difficulty, and perhaps for other reasons, many surgeons have employed two-piece devices, such as those of Thornton and McLaughlin. This appliance is a Smith-Petersen trifin nail fitted with a special locking nut which can be attached at any angle to a lateral plate fixed to the femoral shaft. Mechanically this has inherent weaknesses. It was because he had mechanical failures at the point of attachment of the three-flanged nail with the lateral plate that Kennedy of London, Ontario, began to use the Moe plate in 1945, and in this number of the Journal we publish a report by Kennedy, McFarlane and McLachlin upon 256 fractures so treated. The Moe plate is shaped to the greater trochanter, to the lateral face of which and of the femoral shaft it is fixed, and a firm hold is given to the femoral head and neck by three large lag screws which converge from the outer side of the greater trochanter. One questions whether the apparent greater ease of insertion justifies what looks like a rather massive framework, whether it provides any better mechanical control of the fracture and of intrinsic body forces, and whether the clinical results are any different from those obtained by the use of other devices.

Judged by mortality, Kennedy *et al.* say that the figure of 18·8 per cent " compares well " with rates of Evans and of Scott. Apparently, however, that is a mistaken impression of the comparison, for Evans's mortality for conservative treatment was 15 per cent and for operative treatment 10·9 per cent. Scott's figure for purely conservative treatment was 10 per cent. An assessment of post-operative mortality obtained by Evans from eight published reports of operative treatment (including his own) gave a mean mortality rate of 16·8 per cent. Statistically it is impossible to compare the published mortality rates, because the element of selection, and the time accepted as the post-operative period, are never quite the same. Evans's own criteria were strict, and it is probable that his post-operative mortality might well be regarded as less than 10·9 per cent. In a period of life when morbidity rates are high and mortality is almost a normal incident, one's belief is that operative treatment should not be too critically judged on these rates. Judged by the functional and anatomical results there is little difference between the two methods, though according to Evans there appears to be a somewhat lesser incidence of angular deformity after operation. It is, however, perhaps significant that in the paper by Kennedy *et al.* varus angulation followed the use of the Moe plate in about half the cases, though no accurate figure is given.

The major difference between conservative and operative treatment therefore seems to be in the comfort of the patient and the ease of nursing, for which no statistics can be given, except that for conservative treatment the average length of stay in hospital appears to be about fifteen weeks and for patients operated upon it is five weeks (Evans).

The paper by Kennedy, McFarlane and McLachlin is a careful and modest account of an operative method. Nevertheless if operative treatment is considered advisable, and the present writer is one who believes it is, then a rigid type of nail-plate appears to have superior merit. It should be noted, however, that compared with the report of Evans there is available no considerable study of the results following the use of the two-piece devices of Thornton or McLaughlin.

NORMAN CAPENER.

INCOMPLETE SPINAL CORD INJURIES

With Brown-Séquard Phenomena

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The purpose of this study was to assess the rate and extent of recovery, and the methods of treatment, in a number of incomplete lesions of the spinal cord of the Brown-Séquard type caused by injury. Brown-Séquard defined this condition in the following words: " Spinal hemiplegia is the effect of a disease of, or an injury to, the lateral half of the spinal cord."

The first clear clinical description of this condition, which was derived from a patient treated at the British Hospital at Bahia, was published by Dundas in the *Edinburgh Medical Journal* of 1825. The patient's injury was caused by a fall from scaffolding. However, the first observer to indicate the underlying neurological changes that are responsible for the syndrome was undoubtedly Brown-Séquard.

In 1860 Brown-Séquard in his book, *Physiology of the Nervous System*, described his famous experiment of cord hemisection as follows: " The spinal cord of a mammal is laid bare at the level of the last two or three dorsal vertebrae, and a lateral half of this organ is divided transversely. The animal is left at rest for a little while, and then it is ascertained that sensibility seems to be much increased in the posterior limb on the side of the section, while it seems to be lost, or extremely diminished in the posterior limb on the opposite side . . . (At the same time) there is paralysis on the side of the section, and no paralysis on the opposite side . . . It may be imagined . . . that the conductors of sensitive impressions decussate in the spinal cord, so that those which come from the left side of the body pass into the right side of the spinal cord, and vice versa." He went on to confirm the presence of such decussation by showing that sagittal section of the lumbo-sacral part of the cord in the midline left the hind limbs analgesic although unimpaired in power. It must be noted that strict cord hemisection in man is rare; but it is remarkable how the clinical syndrome in this series of patients resembles the Brown-Séquard pattern of " spinal hemiplegia." Even in closed spinal injuries major damage was restricted to one lateral half of the cord.

TABLE I

NATURE OF INJURY (TWENTY-SEVEN PATIENTS)

Fracture-dislocation of cervical spine	4
Hyperextension injury associated with osteoarthritis of cervical spine	3
Wedge fracture of cervical spine	2
Gunshot wound	16
Wedge fracture of thoracic spine	1
Prolapsed intervertebral disc in mid-thoracic region	1

From time to time in the literature cases of the Brown-Séquard syndrome, due to trauma, have been reported, but as far as we are aware no large series of patients suffering from this condition has been studied. Twenty-seven patients have been examined in this report. The oldest was sixty-nine years of age, the youngest nineteen. The average age of these patients was thirty-two.

The nature of the injury in these twenty-seven patients is shown in Table I. The site of injury in the vertebral column, and in the segments of the spinal cord, is given in Table II. It will be seen that there were sixteen patients with damage to the cervical spine, and eleven with damage to the thoracic spine. All the closed injuries except two occurred in the cervical region. In one patient with a hyperextension injury there was no radiological evidence of

TABLE II

SITE OF LESION (TWENTY-SEVEN CASES)

Number of patients	Site of damage to vertebral column	Initial upper segmental sensory level of spinal cord lesion
2	C.4	C.3 Bilateral C.2 Left
1	C.4–5	C.5 Bilateral
2	C.5	C.5 Bilateral C.5 Bilateral
3	C.5–6	C.4 Bilateral C.8 Bilateral C.8 Bilateral
3	C.6	C.5 Bilateral C.7 Bilateral C.7 Bilateral
1	C.6–7	C.8 Bilateral
1	C.7	C.4 Bilateral
3	No localising evidence	C.4 Bilateral C.5 Bilateral C.8 Bilateral
1	T.1–2	C.5 Left
1	T.3	C.5 Bilateral
1	T.4	T.5 Bilateral
1	T.4–5	T.8 Bilateral
1	T.6	T.7 Bilateral
1	No localising evidence	T.6 Left
2	T.7	T.8 Bilateral T.8 Bilateral
2	T.9	T.10 Bilateral L.1 Bilateral
1	T.11	L.1 Bilateral

damage to the spinal column in the cervical region, but a study of the neurological findings indicated that the lesion had occurred in the region of the fifth cervical segment. Open operation was not performed for any of the closed injuries. In the cases of gunshot wounds the operation was limited to debridement. In three of these patients damage to the spinal cord was observed at operation. Including these three, twelve patients showed radiographic evidence of damage to the vertebral column. No bone damage could be seen on the radiographs of five patients, but metallic foreign bodies on the side of the cord injury, in close relation to the vertebrae, provided evidence of the site of the injury. The nature of the damage to the vertebral column bore no relation to the prognosis.

The lesion affected the left side of the spinal cord in fourteen patients, and the right in thirteen.

Analysis of motor damage—In all patients except one motor damage was greatest at the onset. The exception was a patient with a prolapsed disc in whom the onset was gradual and the first symptoms appeared one hour after the injury at rugby football. In sixteen patients in whom the lesion occurred above the level of the third thoracic vertebra the onset took the form of a flaccid tetraplegia. One patient with a cervical lesion showed only tetraparesis, and one patient with a gunshot wound at the level of the first and second thoracic vertebrae had an initial paralysis of the ipsilateral upper and lower limbs and weakness of the contralateral lower limb, but the contralateral upper limb escaped completely. Nine patients with lesions below the third thoracic segment presented with a flaccid paraplegia.

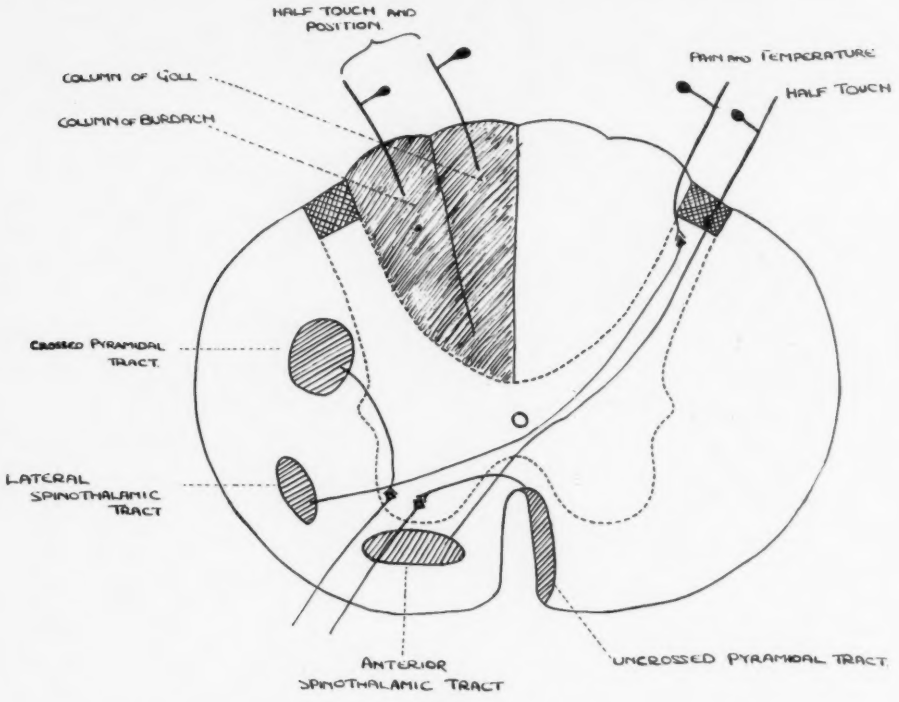

FIG. 1

Diagrammatic cross-section of spinal cord showing position of ascending and descending tracts.

First sign of motor recovery—Some motor recovery occurred rapidly after the onset of the lesion in most patients, being noted earlier on the contralateral side.

The first sign of recovery on the contralateral side was noted as early as twenty minutes and as late as fourteen days in different patients. The average time was fourteen hours after injury if the patient who took fourteen days to show any sign of recovery is excluded, this being an exceptionally long delay. Information is incomplete in three patients. Two patients showed only an initial weakness and one showed no weakness at all.

On the ipsilateral side recovery was first noted between one and fifty-six days, the average period being nine days. The patient in whom recovery was first detected at the fifty-sixth day went on to make excellent progress, being able to walk a mile with the aid of a stick ten years later.

The times considered above are those at which any movement was first observed in a previously paralysed limb, upper or lower. It was impossible to distinguish any fixed sequence of events in the process of recovery. Out of twelve patients with all four limbs affected initially, for whom information is available, recovery began simultaneously in upper and lower limbs both contralateral and ipsilateral in five. In one it began simultaneously in upper and lower limbs on the contralateral side, but in the upper limb before the lower limb on the ipsilateral side. In one it began simultaneously on the contralateral side but in the lower limb before

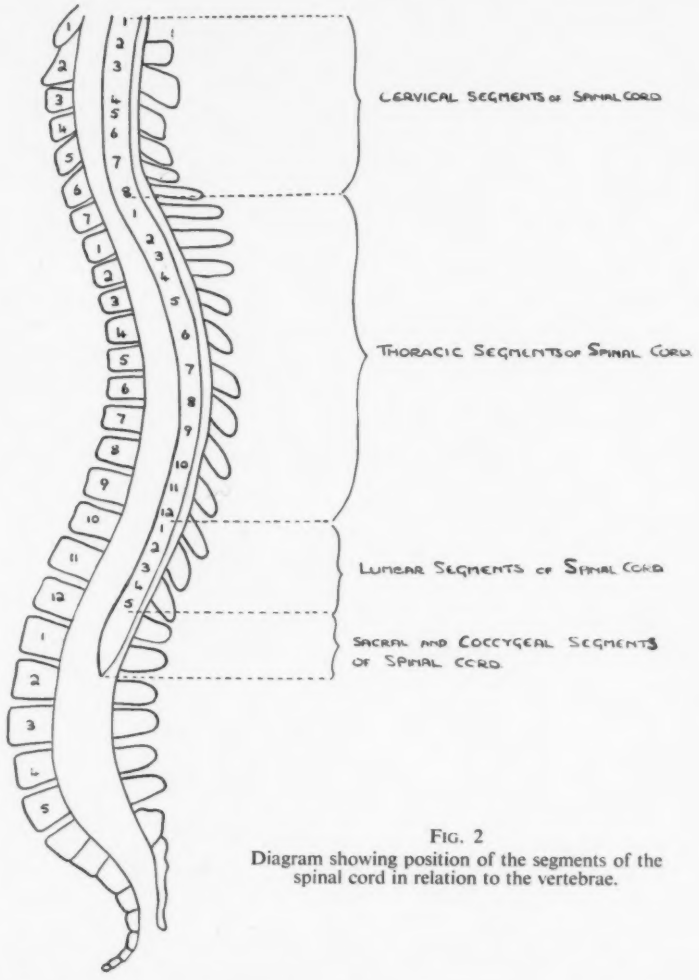
CERVICAL SEGMENTS OF SPINAL CORD
THORACIC SEGMENTS OF SPINAL CORD
LUMBAR SEGMENTS OF SPINAL CORD
SACRAL AND COCCYGEAL SEGMENTS OF SPINAL CORD

FIG. 2
Diagram showing position of the segments of the spinal cord in relation to the vertebrae.

the upper limb on the ipsilateral side. In one it began simultaneously on the ipsilateral side but in the upper before the lower limb contralaterally. In two patients recovery began in the upper before the lower limbs bilaterally. In one patient it commenced in the lower limb before the upper limb on the contralateral side and vice versa on the ipsilateral side. Finally in one patient in whom the two limbs were affected on the ipsilateral side only, recovery began in the upper before the lower limb. When recovery had begun simultaneously the delay varied between ten minutes and six weeks, as shown below.

TABLE III

TIME AFTER INJURY OF FIRST SIGNS OF RECOVERY IN MOTOR AND SENSORY FUNCTION IN PATIENTS WITH INITIAL TOTAL PARALYSIS BELOW THE LESION

The sites of motor recovery and the nature of the changes in sensation are indicated

Case No.	Motor recovery				Change from complete sensory loss			
	Ipsilateral		Contralateral		Ipsilateral		Contralateral	
	Upper limb	Lower limb	Upper limb	Lower limb	Time	Nature of change	Time	Nature of change
1	17 hours	17 hours	17 hours	17 hours	17 hours	Hyperaesthesia. Posterior column loss	17 hours	Dissociated loss
2	1 hour	1 hour	1 hour	1 hour	4 days	Band of hyperaesthesia. Otherwise normal	4 days	Dissociated loss
3	2 days	2 days	2 days	2 days	Insufficient evidence available			
4	6 days	6 days	20 minutes	30 minutes	24 hours	Band of hyperaesthesia. Posterior column loss	24 hours	Dissociated loss
5	2 days	5 days	24 hours	1 hour	1 hour	Hyperaesthesia. Dissociated loss	1 hour	Hyperaesthesia. Dissociated loss
6	8 hours	8 hours	3 hours	3 hours	4 hours	Paraesthesia	4 hours	Paraesthesia
7	30 days	30 days	6 hours	6 hours	2 days	Posterior column loss	2 days	Dissociated loss
8	3 days	14 days	24 hours	24 hours	4 days	Band of hyperaesthesia. Posterior column loss	4 days	Dissociated loss
9	14 days	21 days	—	—	14 days	Band of dissociated loss. Posterior column loss	14 days	Dissociated loss
10	9 hours	27 days	9 hours	12 hours	Insufficient evidence available			
11	—	24 hours	—	8 hours	8 hours	Band of dissociated loss	8 hours	Dissociated loss
12	Insufficient evidence available				Insufficient evidence available			
13	—	56 days	—	14 days	Insufficient evidence available			
14	—	7 days	—	36 hours	Insufficient evidence available			
15	Insufficient evidence available				1 hour	No loss	1 hour	Dissociated loss
16	—	30 minutes	—	20 minutes	30 minutes	Dissociated loss	45 minutes	Dissociated loss
17	—	18 days	—	15 days	4 hours	Increased threshold to all modalities	7 hours	Dissociated loss
18	4 days	66 days	4 days	39 days	108 days	Increased threshold to all modalities	18 hours	Recovery of posterior columns in hallux
							144 days	Dissociated loss
19	2 hours	42 days	2 hours	2 hours	1 hour	Paraesthesia	60 minutes	Paraesthesia

TABLE III—*continued*

TIME AFTER INJURY OF FIRST SIGNS OF RECOVERY IN MOTOR AND SENSORY FUNCTION IN PATIENTS WITH INITIAL TOTAL PARALYSIS BELOW THE LESION

The sites of motor recovery and the nature of the changes in sensation are indicated

Case No.	Motor recovery				Change from complete sensory loss			
	Ipsilateral		Contralateral		Ipsilateral		Contralateral	
	Upper limb	Lower limb	Upper limb	Lower limb	Time	Nature of change	Time	Nature of change
20	—	10 days	—	6 hours	Insufficient evidence available			
21	Insufficient evidence available				Insufficient evidence available			
22	Insufficient evidence available				Insufficient evidence available			
23	3 days	9 days	3 days	3 days	1 day	Increased threshold to all modalities	1 day	Increased threshold to all modalities
24	12 days	33 days	12 days	22 days	—	Posterior column loss	22 days	Dissociated loss
25	Insufficient evidence available				Insufficient evidence available			
26	Insufficient evidence available				Insufficient evidence available			
27	14 days	8 weeks	12 hours	4 days	12 hours	Posterior column loss	12 hours	Dissociated loss

Period of recovery (Table III)—The greatest recovery occurred in the first eighteen months. Rapid onset of recovery is of good prognostic significance. When recovery begins in minutes major recovery occurs within the first two months, though improvement may go on for years.

Recovery on the contralateral side was often complete within a week, but in one patient it continued for as long as eight months.

On the ipsilateral side the lower limb progressed to the stage of what may be described as " paraparesis in extension." A variable amount of voluntary control did recover on this side. Most patients found it an advantage to stand for a short time before walking in order to develop the necessary extensor thrust in the spastic lower limb.

Lower motor neurone recovery—Motor changes at the level of the lesion were largely lower motor neurone in type, from direct damage to the efferent neurones in the spinal cord at the level of the lesion. On the contralateral side recovery started within two days of the date of injury and was complete within two or three weeks, although in one patient improvement continued for as long as eight months.

On the ipsilateral side improvement occurred, but there was a definite neurological deficit of lower motor neurone type in eleven out of sixteen patients with cervical injuries. The remainder showed upper motor neurone type changes. In the former patients the lesion consisted mainly of marked weakness of the muscles supplied by the seventh and eighth cervical and first thoracic nerve roots. Likewise in seven patients there was a band of ipsilateral sensory loss in the eighth cervical and first thoracic segments.

It is interesting that those segments bore the brunt of the damage in over half the patients with injury of the cervical cord, whatever may have been the original neurological level of the lesion. This phenomenon may perhaps be explained by the anatomy of the arteries of Adamkiewicz. According to Bolton (1939) the spinal branch of the first intercostal artery is especially large, and the anterior spinal artery has become very small just above the point where this large lateral vessel joins it. This obviously facilitates a downward rather than an

upward flow of blood, and in injection experiments into the spinal branch of the first intercostal artery this proved to be the case, fluid passing only down the cord. Consequently in any lesion which caused pressure on the anterior spinal artery anywhere between the foramen magnum and the first thoracic vertebra, the parts of the cord most affected by the resulting ischaemia would be those situated farthest from the source of blood flow, that is, the eighth cervical and first thoracic segments.

Onset of spasticity—Spasticity was first noted on the ipsilateral side between three and 123 days, the average period being thirty-two days. In most patients there was some flaccidity at the level of the lesion. On the contralateral side some patients showed no increase in muscle tone, and flaccidity passed into the normal state, but in twelve patients there was some increase in muscle tone which was first noted on the average at thirty-one days. In one patient the contralateral limb was still flaccid at sixty-one days. In general the increase in tone was most obvious in the extensors of the lower limb.

Sensory loss (Table III)—In twenty-one patients there was initial sensory loss of all modalities below the level of the lesion. Three patients showed dissociated sensory loss on first examination. One patient denied any numbness and it was not until eight days had elapsed that dissociated sensory loss was discovered. With regard to two patients full information is not available, and they themselves did not recognise any numbness because they had such severe abdominal pain, presumably due to root irritation. Dissociated sensory loss was discovered in these two patients after five and seven days.

The first sign of recovery in these twenty-one patients was noted between a few minutes and two weeks, the average period being twenty-five hours. Three patients were excepted from this average: in one insufficient detail is known; in the second, recovery did not begin until two weeks had elapsed; and in the third, loss of all modalities persisted for a very long time, except that at eighteen hours this patient could appreciate gross movement of the contralateral hallux and ankle, and further improvement was not noticed until the 108th day on the ipsilateral side. As the Table shows, there is no definite correlation between the onset of motor recovery and of sensory recovery.

The pattern of sensory recovery—On the contralateral side this was a change from complete anaesthesia to dissociated sensory loss, except for those patients in whom loss was dissociated from the beginning. In most patients little further improvement occurred, but in nine a fall in the upper level of sensory loss was noted between five days and eleven months. This fall varied from one to seventeen segments. The average was a fall of eight segments below the level of the lesion. Six of the patients who improved in this way had cervical lesions. One patient with a lower thoracic lesion showed recovery of sacral sensation on the sixth day. Three further patients showed a change from complete to partial dissociated sensory loss at six days, ten months and eighteen months.

On the ipsilateral side the first change from complete anaesthesia was variable. Posterior column loss was noted in twenty patients. Five patients showed a phase of dissociated sensory loss which cleared up completely, except for a narrow band on the upper level of the lesion. Two patients who did not show this phase of dissociated sensory loss retained a band of complete sensory loss at the upper level of the lesion. Patients continued to improve for a variable period. Most had shown the full extent of their recovery within a week, but two continued to improve for one month, four for a year and one for twenty-two months.

The final state of sensory loss—On the contralateral side complete dissociated sensory loss remained in fifteen patients, though there was a falling of level in those already noted; in twelve the dissociated sensory loss became partial.

On the ipsilateral side in seven patients a narrow band of sensory loss remained at the upper level of the lesion. This was a loss of all modalities in two but in five it took the form of dissociated sensory loss.

One patient showed a mild degree of dissociated sensory loss below the level of the lesion on the ipsilateral side at eight months, but he died at one year from another cause and it is possible that further improvement might have occurred.

Posterior column loss remained complete in three patients, became incomplete in ten, and disappeared in the remaining seven.

Hyperaesthesia—This was a marked feature in fifteen patients. The onset varied from the time of injury to 123 days, the average period being twenty-four hours, if three patients are excluded. In these latter the condition was noted at 108, 120 and 123 days. The zone of hyperaesthesia coincided with the upper level of the neurological lesion, and spread to a variable extent on either side of it. It was always present on the ipsilateral side, though in six patients it was bilateral. Its average duration was thirty days, but in one patient it passed off in three days and in four patients it was still troublesome years later.

Root pain—Spontaneous pain of root distribution occurred in fifteen patients. It was of immediate onset in four patients. It led to an exploratory laparotomy in one patient and in another to the clinical suspicion of a ruptured intra-abdominal viscus. The distribution of the root pain was that of the upper level of the neurological lesion. When the onset was not immediate it varied from four hours to thirteen months, the average period being twenty-eight days, if this last patient is excluded.

TABLE IV

TIME RELATIONS OF HYPERAESTHESIA AND ROOT PAIN IN PATIENTS SUFFERING FROM BOTH

Patient	Onset		Persistence	
	Hyperaesthesia	Root pain	Hyperaesthesia	Root pain
1	17 hours	Immediate	5 years	33 days
2	4 days	Immediate	Still present	6 days
4	6 days	24 hours	Still present	Still present
5	Immediate	Immediate	20 days	12 days
6	15 hours	4 hours	3 days	3 days
8	3 days	3 days	4 months	Still present
11	8 hours	40 days	31 days	188 days
12	4 months	Immediate	30 days	7 days
13	24 hours	13 months	7 years	6 years
16	Immediate	Immediate	1 year	1 month
20	Immediate	Immediate	54 days	Still present

Root pain tended to be bilateral and in only three patients was it unilateral. There was recovery from root pain in eleven patients at an average period of sixty-four days from the onset, if two are excluded in which the pain lasted five and seven years respectively before it ceased. It persisted in three patients throughout the period of follow-up.

The eleven patients with root pain and hyperaesthesia coincided but in four there was root pain without hyperaesthesia, and in four there was hyperaesthesia without root pain. When both phenomena were present together time relations were by no means identical (Table IV).

Paraesthesia—Out of ten patients with cervical and eleven with thoracic injuries about whom information is available, paraesthesia was present in eight of the former, but only in two of the latter. The onset was within twelve hours except in two patients, one with a cervical injury

in whom it was noted at ten days, and one with a thoracic injury in whom it began at fourteen days. In six patients, all with cervical injuries, it was a generalised phenomenon; in one cervical injury it was confined to both upper limbs and in another to the contralateral upper limb. In one thoracic injury it affected only the contralateral lower limb, and in the second it was felt partly in both lower limbs. It persisted on the average for four days after the onset, with two exceptions—both cases of cervical injury—in which it lasted in a localised form for two and four years.

Tendon reflexes—There was an initial loss of all tendon reflexes in the affected limbs. On the contralateral side the reflexes returned fairly rapidly in a period varying from a few hours to several days depending on the severity of the injury. They then remained normal or showed an increase depending on the degree of spasticity present.

On the ipsilateral side the return of tendon reflexes took up to three weeks. There was marked increase in the lower limbs with clonus. There was also marked increase in the upper limbs except where the lesion was of the lower motor neurone type. These defects have persisted over the period of follow-up.

Cutaneous reflexes—These were absent initially, and again their return was variable. On the contralateral side the onset of recovery varied between eight hours and several days. A plantar reflex was always obtained within three days, usually earlier, and was with one exception upgoing. In this latter patient the response was normal in three days. The plantar response eventually became normal in two-thirds of the patients within periods varying from four days to one year, the average period being thirty days. The abdominal reflexes usually remained absent until the plantar response became normal, when they too became normal; but in two patients it was possible to elicit a flicker before any plantar response appeared.

On the ipsilateral side a longer period elapsed before the plantar reflex could be obtained. It then became upgoing and remained so during the period of follow-up. The abdominal reflexes remained depressed or absent. Two patients showed complete recovery at five months and eleven years respectively.

Disturbances of micturition—Urinary function was unaffected in six patients. It is difficult to account for this because all except one of these patients had extensive motor and sensory damage. Three had cervical, and three thoracic, lesions. The patient with slight motor and sensory changes had a lesion of mild degree at the eighth thoracic segment.

Three patients had difficulty in initiating the flow but none required treatment. One made a complete recovery, one was left with nocturnal frequency, and one with slight difficulty in initiating the flow. One patient with incontinence made a complete recovery but in one patient urgency persisted.

Seventeen patients had immediate retention which required treatment. Five of these had suprapubic drainage which was removed between three weeks and three months, the average being one month. Eleven had tidal drainage which was maintained until control of micturition developed. Of all these, thirteen developed normal control of their bladder, one developed an automatic bladder, one had some hesitancy in commencing micturition, and one had precipitancy and nocturnal incontinence which compelled him to use a urinal at night.

The only death in the series occurred in the patient who had the suprapubic cystostomy maintained for three months before attaining control of his bladder. The same gunshot wound that caused his spinal injury also severely damaged a kidney, which was excised. As a result of chronic urinary infection he developed nephrolithiasis in the remaining kidney and died of uraemia a year after the injury.

Sexual function—Information regarding sexual function was available in twenty-one male patients. Eleven were unaffected, nine were impotent, and one had impaired function in the initial stages. Six of these nine patients remained impotent. Two recovered, one at one year, one at two years. Although erections were absent in two patients, emissions were obtained with the aid of testosterone.

Bowel function—Eight patients had normal bowel function. Seventeen patients had retention of faeces and two had incontinence. Seven of these patients had made a complete recovery by the end of six months, but eleven still complained of constipation, and one of these, who had been incontinent initially, had to empty his bowel in anticipation in order to avoid accidents. In general, bladder, bowel and sex dysfunction went hand in hand, but one patient had complete retention of urine and an unaffected bowel, and four patients who had bladder and bowel disturbances were not affected sexually.

Horner's syndrome—This syndrome occurred in seven of the cervical and one of the upper thoracic injuries, all being caused by gunshot wounds. The condition was absent in the closed injuries. Recovery took place in all patients over periods varying from twelve days to eight years.

Lumbar puncture—Lumbar punctures were performed on seventeen patients. When this was done in the first week a moderately blood-stained cerebro-spinal fluid was found. After the first week the findings were normal. In two cases there was a partial block in the initial stages, but this had cleared spontaneously by the forty-eighth and sixty-ninth day.

TREATMENT

Treatment of the primary lesion—The injuries have been subdivided into two groups: 1) Those caused by gunshot wounds; and 2) those associated with closed injuries to the vertebral column.

The sixteen patients who received gunshot wounds were treated by surgical excision and removal of foreign bodies in field surgical units, or casualty clearing stations, and were then transferred to base hospitals in the United Kingdom for further treatment.

The eleven patients with closed injuries of the spine were treated in the following manner.

1) Four patients had fracture-dislocations of the cervical spine. These dislocations were reduced by skull traction. Skull traction was maintained for an average of six weeks, and then the patients were immobilised in plaster jackets of the Minerva type. These were worn for twelve weeks on an average.

2) Two patients sustained wedge fractures of the cervical spine. These fractures were reduced by postural extension, and the patients were immobilised in Minerva plasters for twelve weeks.

3) In patients with hyperextension injuries and osteoarthritis of the cervical spine reduction by gentle flexion was followed by immobilisation in a Minerva plaster for twelve weeks.

4) One patient had a wedge fracture of the mid-thoracic spine. He was nursed in slight extension with a pillow under the mattress for eight weeks.

5) One patient showed on myelography what appeared to be a prolapsed intervertebral disc between the fourth and fifth thoracic vertebrae. He was kept in bed in the initial stages, but the paresis and loss of sensation were slight, and he was able to resume relatively normal life within a month of getting up. He was, however, invalided from the Army as medically unfit.

The degree of respiratory paralysis depended upon the site and severity of the lesion, but patients with cervical and high thoracic injuries were at first solely or largely dependent on diaphragmatic movement to maintain their respiratory exchange. It was wise, therefore, to nurse these patients in the horizontal position or with the foot of the bed slightly raised, so that the weight of the abdominal viscera provided some expiratory force. Fortunately recovery in these patients was much more rapid than that which occurs in anterior poliomyelitis, and lying on the back was therefore not so dangerous. From this point of view one patient was severely affected, and he was turned two-hourly to promote lung drainage as well as to protect the skin. This was done with great care and under the supervision of a doctor until the nursing staff were thoroughly conversant with the precautions to be taken. The condition of the lungs must be kept under careful observation, and breathing exercises instituted as far as possible.

Management of the sphincters—Urinary retention requiring treatment occurred in seventeen patients. Seven were subjected to suprapubic cystostomy. These patients were all battle casualties, the operation being made necessary by the fact that they had to be transferred back along the lines of communication. Under more settled circumstances the patients were treated by intermittent catheterisation and tidal drainage if necessary. The intermittent catheterisation was carried out twice or thrice daily for seven to fourteen days. If micturition had not returned at the end of this time, tidal drainage was instituted with a Foley's indwelling catheter, changed every five days. All these procedures were carried out under the strictest possible aseptic precautions. Urinary infection was minimal, but as an additional safeguard about half the patients were given doses of 0·5 gramme of sulphamezathine eight hourly.

With regard to the bowel, constipation was the major problem in most patients, and proved to be troublesome in thirteen. In the initial stages enemata and glycerine suppositories were employed. After a few weeks all that was usually necessary was an occasional mild purgative. In one patient digital evacuation had to be taught, and in two patients in whom the occurrence of defaecation could not be felt pads of wool were worn for protection.

Muscle rehabilitation—As soon as the patient's general condition allowed (usually two to six weeks after the injury) rehabilitation in walking was commenced in the pool.

One of the most distressing features of this condition was the presence of involuntary flexor and extensor spasms which occur with greatest severity in the lower limb on the affected side. In addition increased muscle tone produces cramp-like pain from time to time. In treatment, spasticity is the most difficult problem to be faced. The use of antispasmodic drugs has proved to be of little value, and in fact there is no known method of controlling such phenomena, except by destruction of the motor nerves. The only way to reduce spasticity is to avoid all abnormal or excessive stimuli both internal and external. Excessive cold and damp should be avoided, the condition of the bowel should be adequately controlled and the bladder emptied especially before going to bed at night. Warm pool therapy, when the condition of the sphincters allows it, is of great value because it gives the patient a feeling of complete relaxation, and helps him to walk by reducing the force of gravity. Standing on the floor should also be attempted in order to develop the extensor thrust in the leg on the affected side. After the first week's pool therapy walking should be practised with the aid of parallel bars. It is helpful to arrange a mirror, so that the patient may visualise the position of the affected limb when the posterior column is involved. If flexor spasms are severe it is sometimes helpful to nurse the patient in the prone position for short periods. It is of value if the patient stands for a few minutes with his weight on his spastic limb before attempting to walk, to develop an adequate extensor thrust. Walking is taught between the parallel bars; elbow crutches are required at first, but eventually the patient can usually walk with sticks or even unaided.

Active exercises are required for the upper limbs, and occasionally, in the more severely affected, wax baths are used to promote the restoration of hand function. In spite of treatment however, the ipsilateral hand of eight of the patients with cervical injuries can be described as virtually useless.

Protection of the analgesic part—As a result of damage to the spino-thalamic tracts, the protective influence of pain is lost. It is therefore important that the analgesic areas of the trunk and lower limbs should be protected from burns with hot water bottles, and that the individual himself should be taught to protect such areas from injuries of all kinds. It is obvious that the pressure areas should be carefully watched during treatment in recumbency.

PATHOLOGY

No certain knowledge of the nature of the damage to the cord in these patients exists, because all except one are still alive. No examination of the spinal cord was made in the patient who died a year after the injury. In three patients the meninges were exposed at the

time of wound excision, and bone fragments were seen pressing on the cord, though the dura appeared intact.

It may be inferred from the clinical course of these patients that the pathological changes were concussion, oedema and haematomyelia, as well as some direct structural damage of the cord. Loss of function, from which recovery occurs in a matter of minutes or a few hours, must be due to concussion. The disappearance of oedema and the resolution of haemorrhages are presumably responsible for the improvement occurring within the first ten days and three months respectively.

PROGNOSIS

Motor recovery was rapid on the contralateral side, but was much slower on the ipsilateral side, where it remained incomplete, with spastic paresis in extension, but with some retention of voluntary control. Involuntary movements of a spastic nature remain a distressing feature on this side.

Major recovery occurred during the first eighteen months, but goes on for years. There is no fixed sequence of events. The more rapid the recovery the better the prognosis.

There was initial total sensory loss which passed rapidly into dissociated sensory loss. There was no correlation between the onset of sensory and of motor recovery. Hyperaesthesia was present at the upper level of the lesion on the ipsilateral side; occasionally it was bilateral, and it usually passed off. Root pain also occurred at the upper level of the lesion, and could simulate an intra-abdominal catastrophe. It tended to be bilateral.

Tendon reflexes returned rapidly on the contralateral and more slowly on the ipsilateral side, as did cutaneous reflexes.

Recovery of urinary function took place during the first six weeks. Rather less than a quarter of the patients were rendered impotent. Horner's syndrome, when present, was only transient.

Sixteen patients were followed up for between six and eleven years, seven for between three and four years, and three for two years. One patient died at one year from uraemia as an indirect result of his injury. At the time of his final illness he had progressed far enough to walk with sticks.

The most severely injured patient in this series was walking with the aid of elbow crutches and beginning to use a typewriter at two years. The remainder are all earning their own living, eleven being in protected employment as partly disabled persons. Eight patients require a stick or sticks for walking. At the end of the follow-up period all except one could walk over a mile and many much farther.

In eight of the patients with cervical injuries the ipsilateral hand was virtually useless.

SUMMARY

1. Twenty-seven patients with a Brown-Séquard syndrome resulting from trauma have been studied, fourteen of the left side of the cord and thirteen of the right. There were sixteen gunshot wounds and eleven closed injuries.

2. The prognosis for recovery is much better than the initial catastrophic nature of the symptoms and signs would indicate.

3. The pattern of recovery is discussed in detail and the long and rather tedious course of the treatment is indicated.

4. Spasticity on the side worse affected still presents a difficult problem, but a less severe one than that presented by flaccid paralysis.

REFERENCES

BEDFORD, P. D., BOSANQUET, F. D., and RUSSELL, W. R. (1952): Degeneration of the Spinal Cord Associated with Cervical Spondylosis. Lancet, **ii**, 55.

BOLTON, B. (1939): The Blood Supply of the Human Spinal Cord. Journal of Neurology and Psychiatry, **N.S. 2**, 137.

BROWN-SÉQUARD, C. E. (1860): Course of Lectures on the Physiology and Pathology of the Central Nervous System. Philadelphia: Collins.
BROWN-SÉQUARD, C. E. (1868): Lectures on the Physiology and Pathology of the Nervous System; and on the Treatment of Organic Nervous Affections. Lancet, **ii,** 593, 659, 755, 821.
DUNDAS, R. (1825): Case of Concussion of the Spine, Tending to Confirm the Opinion, that the Nerves of Sensation and of Motion are Distinct. Edinburgh Medical Journal, **23,** 304.
GUILLAIN, G., and GARCIN, R. (1931): Le Syndrome de Brown-Séquard d'origine traumatique. Annales de Médecine, **29,** 369.
GUILLAIN, G., and CORRE, L. (1950): Considérations sur un cas de syndrome de Brown-Séquard d'origine traumatique exceptionnelle. Bulletin de l'Académie Nationale de Médecine, **134,** 147.
JEFFERSON, M. (1952): Brown-Séquard. Lancet, **i,** 760.
JEFFERSON, M. (1954): The Brown-Séquard Syndrome. Medicine Illustrated, **8,** 209.
LAST, R. J. (1956): Personal communication.
RUSSELL, W. Ritchie (1952): Personal communication.
RUSSELL, W. Ritchie (1954): Poliomyelitis. *In* British Surgical Practice. Surgical Progress 1954, p. 147. London: Butterworth & Co. (Publishers) Ltd.

THE MOE PLATE IN INTERTROCHANTERIC FRACTURES OF THE FEMUR

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The plate designed by Dr J. H. Moe of Minneapolis for internal fixation of intertrochanteric fractures of the femur has been mentioned in the literature only twice (Lamphier, Trott and Shortell 1948, Kennedy 1950). It constituted only 1 per cent of the appliances for internal fixation of this fracture sold by a major United States surgical supply house in 1954.

We began to use the Moe plate in 1945 because of mechanical failures at the junction of the three-flanged nail and lateral plate that we were then using. Our continuing satisfaction with the method seemed to warrant bringing it to further attention.

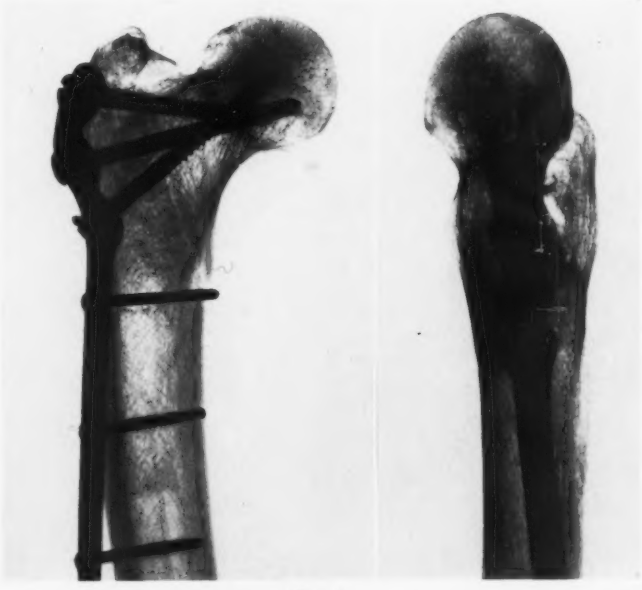

FIG. 1
Radiographs of a specimen removed one month after internal fixation of an intertrochanteric fracture by a Moe plate.

The plate and its purpose are shown in radiographic studies of a specimen recovered one month after operation (Fig. 1). The upper end of the plate is curved to fit the greater trochanter. Through this upper end three large lag screws inserted into the neck and head in converging directions afford a strong hold on the proximal fragment and also approximate the fracture line. The lower blade has three or four holes permitting fixation to the shaft of the femur. The various plate sizes usually allow a good fit without bending the plate. The apparatus is structurally strong, and in comminuted fractures in which perfect reduction is not obtainable the screws can be placed to best advantage.

In the period from May 1945 to December 1954, 254 out of 270 patients with intertrochanteric fractures of the femur were treated with a Moe plate (Table I).

In two patients marked comminution made it impossible to obtain secure fixation of the fragments and the attempt was abandoned in favour of traction. In two others a very long McLaughlin plate was used because the fracture ran far enough down the shaft of the femur to be beyond the limits of the longest Moe plate.

TABLE I

TREATMENT OF 270 INTERTROCHANTERIC FRACTURES OF THE FEMUR

Moe Plate	254
Attempted plating	2
McLaughlin plate	2
Not operated upon	12

Of the twelve patients not operated upon only four survived. These included three with crack fractures that required only rest in bed. The remainder of the twelve were all very poor subjects. Three were admitted in extremis and died soon after. If the three patients who required neither operation nor traction are excluded, there was thus only one survivor in the group treated by a closed method. The bad results in these very poor subjects must not of course be compared with the operative results. The deaths are included in the overall series. In this follow-up no hospital deaths in patients admitted with intertrochanteric fractures have been excluded irrespective of length of time or apparent irrelevancy of the fracture to the death (Table II).

TABLE II

HOSPITAL MORTALITY

Mortality	Number of patients	Deaths	Mortality
Overall	270	56	20·7 per cent
Operative	256	48	18·8 per cent
Non-operative	12	8	66·6 per cent

INTERTROCHANTERIC FRACTURES TREATED WITH A MOE PLATE

The remainder of this paper will be confined to a study of the 256 cases in which an attempt was made to use a Moe plate and will include the two cases in which the attempt was given up in favour of traction.

The average age of 75·2 years supports the view of Lamphier, Trott and Shortell (1948) that intertrochanteric fractures occur in an older age group than fractures of the femoral neck. The influence of age is shown by the fact that the survivors had an average age of 74·1 years and those who died an average age of 80·1 years.

These cases have not been considered surgical emergencies, and we believe that the advantages of operating with a daytime staff outweigh any gain in time by operating at night. Medical consultation has been obtained in all debilitated patients and, if it seemed best, operation was postponed. We believe that in some instances a few days of intensive medical management are of more value than early operation. Because of the malnutrition and contracted blood volume so common in the poorer subjects, we have frequently given whole blood before operation. With the exceptions described above we have, by preference, carried out operation within twenty-four hours of admission to gain the benefits of early mobilisation of the patient. The average time from fracture to admission was 1·4 days (0–46) and from fracture to operation 3·0 days (0–47). Adhesive traction in a Thomas's splint or with the support of sandbags has been used pending operation.

The operation itself requires little description beyond emphasis on the co-operation between surgeon and x-ray staff that can be so time saving. Radiographs of an initial guide wire passed through one of the upper plate holes into the head permit insertion of lag screws of accurate length and direction. A well planned and executed procedure should not require more than one hour. In the more debilitated subjects even small blood loss seems poorly tolerated and 500 millilitres of blood have usually been given during the operation. Expert anaesthetists were available and the choice of agent was left to them. General anaesthesia was most commonly used.

After operation every effort was made to get the patient out of bed early, and most were lifted into a chair on the first day after operation. No harm occurred and, in addition to increasing pulmonary ventilation and relieving skin pressure, the practice has great value in lessening the despair so often seen in old people confined to bed for a long time.

Suitable patients were encouraged to walk with crutches after six or eight weeks but, in the majority, balance was inadequate for safety until union had occurred at about three months. The reduction in hospital time as compared to traction methods is shown by the survivors' spending an average of forty-seven days in hospital.

MORTALITY WITH THE MOE PLATE

The mortality rates shown in Table II are, as stated above, based on all deaths occurring in hospital irrespective of length of time or appearance of new disease after admission with an intertrochanteric fracture. The overall rate of 20·7 per cent is discouraging but compares well with others reported (Evans 1951, Scott 1951). The operative mortality of 18·8 per cent seems high but must be considered in the light that almost all patients were operated on in hope of decreasing the overall mortality in an old and debilitated group.

About a third of the deaths occurred within ten days after operation and another third by the end of thirty days. It seemed probable that fracture and operation should be considered the major cause of death in this group. In the remaining third death occurred between one month and six months after operation; in these cases age and general condition might be considered more important than the fracture and operation.

TABLE III

CAUSE OF FORTY-EIGHT HOSPITAL DEATHS

Bronchopneumonia	15
Cardiovascular disease	10
Cerebrovascular disease	5
Pulmonary embolus	5
Sepsis	4
Post-operative shock	1
Subdural haematoma	1
Intestinal obstruction	1
Unknown	6

As might be expected, thirty-five of the forty-eight deaths were due to pulmonary, cardiovascular or cerebrovascular disease (Table III). Of the four patients who died from sepsis, two were diabetic and one of these could not be controlled from the time of operation. All four had extensive areas of decubitus ulceration, and in the two that came to necropsy there was extensive pelvic and retroperitoneal suppuration. The patient who died from shock was early in the series and received no blood: this should be considered an error in management.

Complications—Decubitus ulceration of some degree was very common and, though usually superficial and responding to treatment, has been recorded in every case to emphasise its importance. Seventy-five per cent of the ulcers were over the sacrum, and ulceration was multiple in half the cases in which it occurred. Nine patients were admitted with decubitus

ulcers, but in the remainder the average time from operation to skin breakdown was eleven days. This suggested that pressure on the operating table was not responsible, but rather that patients were spending too much time in the dorsal decubitus position in spite of strenuous efforts at turning and skin care.

TABLE IV

POST-OPERATIVE COMPLICATIONS WITH THE MOE PLATE

Decubitus ulcer	45 per cent
Wound infection	6 per cent
Thrombophlebitis. . . .	4 per cent

Many of these skin lesions seemed at least in part due to the urinary incontinence so often seen after the operation. In addition to using flannelette sheets to lessen the abrasive effects of the ordinary hospital linen, we now insert an indwelling Foley catheter at the first appearance of urinary incontinence.

Securing sufficient food and fluid intake in these elderly patients who sometimes resist all attempts to help them can be a difficult problem. We now make almost routine use of nasal feeding through a fine vinylite tube left continually in position in patients who will not voluntarily accept an adequate intake. " Boxing glove " dressings may be as effective as restraining straps and less disturbing to the patient. By these means, some patients who at first seem too disorientated to survive may be carried through to a surprising return of their mental faculties and a subsequent good recovery.

Most wound infections were trivial and associated with skin sutures; these had been left in ten to fourteen days so that patients might be rolled to either side. All have been included. Once again urinary incontinence was often associated with wound infection. The plate and screws required removal because of infection in two instances.

Thrombophlebitis occurred in 4 per cent of cases and was always on the injured side.

RESULTS

Of the 256 patients, 161 or 63 per cent were followed adequately for classification. Of the remainder, forty-eight had died in hospital and forty-seven were lost because of death out

TABLE V

NUMERICAL EVALUATION OF RESULTS WITH THE MOE PLATE

Anatomical result		Functional result	
Grade	Per cent	Grade	Per cent
1	8·8	1	11·1
2	19·7	2	20·1
3	26·5	3	32·5
4	44·9	4	36·4
Average grading 3·1		Average grading 2·9	

of hospital or inaccessibility. The shortest time between fracture and assessment was one year. Patients confined to bed or a wheel chair before operation were not given a functional grading. The results are shown in Table V.

The following criteria were used in the grading:

Anatomical—1) Severe malunion; 2) union in poor position; 3) union in good but not perfect position; 4) union in perfect position.

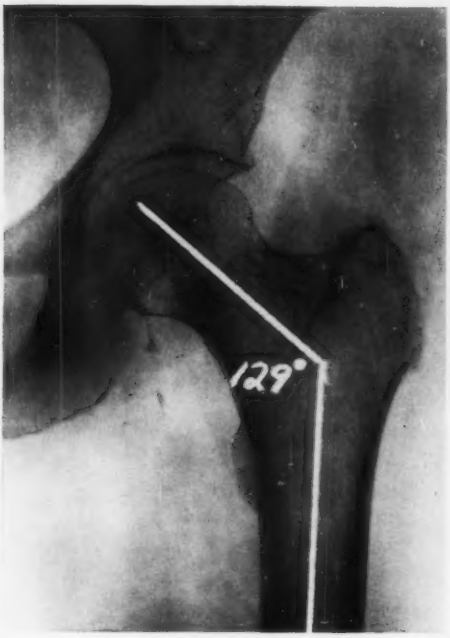

FIG. 2

FIG. 3

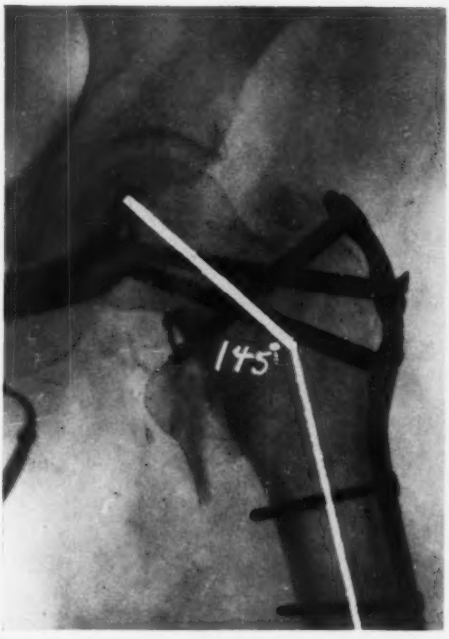

FIG. 4

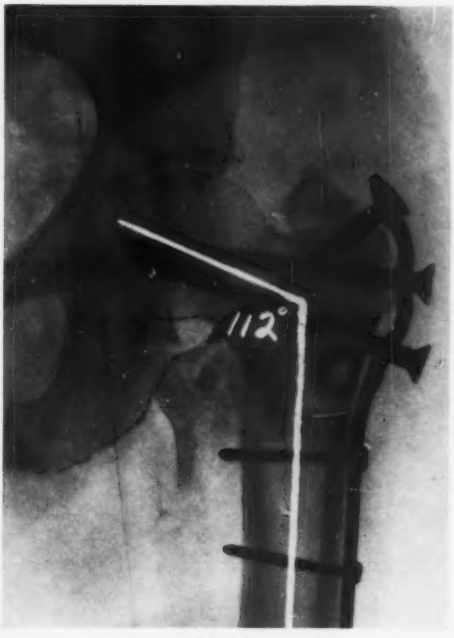

FIG. 5

Radiographs showing the development of varus angulation despite the fact that the patient was not bearing weight on the limb. Figure 2—Before fracture. Figure 3—The fracture before internal fixation. Figure 4—During internal fixation by Moe plate. Figure 5—Three months after operation.

Functional—1) Able to walk before fracture; bedridden or confined to a wheel chair after fracture. 2) Able to walk before fracture; required crutches or stick constantly after fracture; able to walk short distances only; persistent pain; severe gluteal lurch. 3) Minor disabilities after operation such as occasional need of a cane or crutch; some pain or slight limp. 4) Walking as well after fracture as before.

The average achievement of about grade 3 on both anatomical and functional assessments seemed fairly satisfactory in this aged group. Those patients in grades 1 and 2—a total of 31·2 per cent—might be considered as unsatisfactory, and those in grades 3 and 4—a total of 68·9 per cent—as satisfactory functional results. The commonest complication in the survivors who had obtained union and a reasonably good result was a tendency to varus deformity. This has been described by Evans (1951) and by Taylor, Neufeld and Nickel (1955) and occurs after non-operative as well as after operative treatment. Early weight bearing

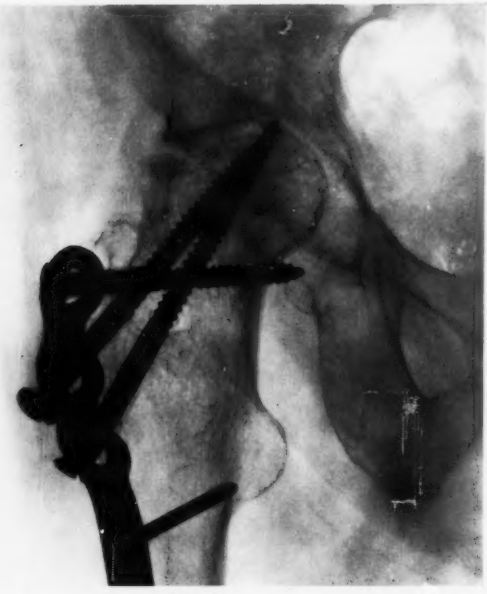

FIG. 6
Avascular necrosis of the femoral head after an intertrochanteric fracture.

may be a factor but it is not entirely responsible, as shown in Figures 2 to 5. The woman in this case had been radiographed for another reason before the fracture. A Moe plate was applied two days after the fracture in purposeful valgus because of the known tendency to varus drift. Radiographs taken three months after fracture show the marked varus deformity although she had borne no weight.

Because of the inaccuracy of measuring femoral neck angles in standard radiographs we have been unable to determine the exact incidence of varus deformity in this series, but we believe that it has occurred to a significant degree in about half the cases. It is compatible with a good functional result.

Traction after operation was necessary in 3 per cent of cases with severe comminution that made it impossible to secure stable internal fixation. With increasing experience we have come to believe that some intertrochanteric fractures are unsuitable for any form of internal fixation and should be treated by traction. Large intermediate fragments including the lesser

trochanter, especially in combination with vertical fractures running well down into the femoral shaft, are particularly difficult. A careful study of good lateral views as well as antero-posterior projections may give warning in these unsuitable cases.

Evans (1949) stated that non-union was rare and avascular necrosis of the femoral head did not occur in intertrochanteric fractures. Taylor, Neufeld and Nickel (1955) reported one avascular necrosis in a series of over 3,000 cases. In this series there was one case of non-union that became evident with a stress fracture of the plate at eleven months. Bone grafting was carried out successfully. The single instance of avascular necrosis (Fig. 6) was an unexpected finding at follow-up four years after fracture.

With development of the varus deformity some extrusion of the screws often occurs. This has produced little disability and has warranted removal of the plate and screws in only 3 per cent of the survivors and of a single screw in another 1 per cent. There has been a stress fracture of a single plate, as described above, and of one lag screw.

SUMMARY

1. The Moe plate has been used in 256 of 270 consecutive intertrochanteric fractures of the femur. There was an operative mortality of 18·8 per cent and an overall mortality of 20·7 per cent in a group averaging 75·2 years of age.

2. The method proved satisfactory, giving secure fixation and also approximation of the fragments by the lag action of the three large screws driven into the head. There was only one case of non-union.

3. In the survivors anatomical and functional results have been satisfactory in about 70 per cent, with half of these showing full functional recovery and the other half requiring only occasional use of a stick or crutch.

4. There are a few intertrochanteric fractures of the femur too comminuted for any form of internal fixation. Traction should be used in this group.

5. A varus deformity developed in about one half of the cases. This is consistent with a good functional result.

6. The main difficulty in these aged people has been survival. The problems have been discussed with suggestions that may perhaps reduce in part a mortality that seems inherent in this injury and at this age.

REFERENCES

EVANS, E. M. (1949): The Treatment of Trochanteric Fractures of the Femur. Journal of Bone and Joint Surgery, **31–B,** 190.

EVANS, E. M. (1951): Trochanteric Fractures. Journal of Bone and Joint Surgery, **33–B,** 192.

KENNEDY, J. C. (1950): The use of the Moe plate in intertrochanteric fractures of the femur. Journal of Bone and Joint Surgery, **32–B,** 752.

LAMPHIER, T. A., TROTT, A. W., and SHORTELL, J. H. (1948): The Use of the Moe Plate in the Treatment of Intertrochanteric Fractures. Surgery, Gynecology and Obstetrics, **87,** 652.

SCOTT, J. C. (1951): Treatment of Trochanteric Fractures. Journal of Bone and Joint Surgery, **33–B,** 508.

TAYLOR, G. M., NEUFELD, A. J., and NICKEL, V. L. (1955): Complications and Failures in the Operative Treatment of Intertrochanteric Fractures of the Femur. Journal of Bone and Joint Surgery, **37–A,** 306.

CONGENITAL DEFORMITY OF THE CARPUS ASSOCIATED WITH MALDEVELOPMENT OF CERTAIN THENAR MUSCLES

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In 1943 Hodgson described a case of " congenital retardation in development of the carpal navicular, first metacarpal and styloid process of the radius " in association with wasting of the thenar muscles and recurrent sprains of the wrists. He was unable to find any previous description of a similar case and no other seems to have been recorded since 1944. The present paper describes two further patients with this condition, each of whom had additional congenital abnormalities.

CASE REPORTS

Case 1—A woman of thirty-one was admitted primarily for mitral valvotomy, and attention was drawn to the deformity of her hands by the finding that arterial pulsation at the wrists was greatly diminished. Both hands had a characteristic rather simian appearance (Fig. 1). There was marked radial deviation at the wrists, the movements of which were, however, painless and of normal range. The thumb was abnormally long and slender and resembled an extra finger, this appearance being accentuated by the almost total absence of the thenar muscular eminences. The power of abduction of the thumb (at right angles to the palm), of opposition of the thumb and of flexion of the first metacarpo-phalangeal joint was greatly reduced, and the muscles concerned—abductor pollicis brevis, opponens pollicis and flexor pollicis brevis—gave a negligible response to faradic stimulation. On the other hand the adductor pollicis was present and of normal strength, as were the first and other interosseous muscles. The hypothenar muscles were present, though slightly underdeveloped. There was no apparent wasting of the muscles of the forearm or upper arm. The supinator, biceps and triceps reflexes were present and equal on the two sides, and there was no loss of sensation. Systemic neurological examination revealed no abnormal signs. Arterial pulsation could not be felt in either radial artery, and only a very feeble pulsation was detectable in the ulnar vessels, whereas brachial and axillary pulsation was easily felt. Arterial pulsation at the wrist was not increased by elevating the arm. Nothing abnormal was palpable at the root of the neck.

The patient said that her hands had been " queer " all her life, but that they had caused her little disability apart from slight weakness of the grip. She had not suffered any injury to the wrists, and had not been subject to sprains. She had had seven children, none of whom is, so far as she can tell, similarly affected (they were not examined by us), and she knows of no comparable case amongst her relations.

Radiological examination. Left hand—The scaphoid bone was strikingly smaller than normal and had a sharply defined elliptical shape quite unlike the normal scaphoid (Fig. 2). The density of the bone was slightly increased. The proximal end articulated with the lateral aspect of the articular surface of the radius, but a wide gap separated the distal pole from the capitate and trapezium. The styloid process of the radius was poorly developed, the articular surface

FIG. 1

Case 1—Right hand shown with a normal hand for comparison. The hand and arm of the patient were in the most comfortable position; the normal hand was not.

FIG. 2

Case 1—Radiographs of the hands.

of the lower end of the radius being set at right angles to the long axis of the shaft, so that the ulnar and radial styloids lay at the same level. The base of the first metacarpal bone was rounded and showed neither an abductor tubercle nor the customary saddle-shaped articular surface. In consequence the first carpo-metacarpal joint was very shallow. The shaft of the first metacarpal bone was unusually elongated and slender. A single sesamoid bone was present at the first metacarpo-phalangeal joint. *Right hand*—In this hand no separate scaphoid could be identified but the articular surface of the radius was irregular and the appearances suggested that a deformed and rudimentary scaphoid was joined with the lower end of the radius (Fig. 2). In addition there was fusion of the capitate and hamate bones. The first carpo-metacarpal joint was shallow, and the first metacarpal bone was unduly long and thin, as in the left hand. *Feet*—Both feet showed conjunction of the cuboid and lateral cuneiform bones. *Thoracic inlet*—Large bilateral cervical ribs were present.

Case 2—A man of sixty-three presented with a complaint of pain in the legs and difficulty in walking. In the course of neurological examination his hands were noted to have a curious simian appearance and there was almost complete absence of the thenar eminences. There was, however, no wasting of either the first interosseous or adductor pollicis muscles and the other intrinsic muscles of the hands also appeared normal. In spite of the apparent absence of the opponens pollicis muscle his grip was fairly strong. Neurological examination showed no other abnormality: the deep reflexes of the arms and legs were normal and there was no sensory loss. He said that the deformity of his hands had been noticed in early

FIG. 3
Case 2—Radiographs of the wrists.

childhood and that it had caused him no disability, although he had worked as a labourer for many years. He had never had pain in the arms or shoulder girdle, nor had he ever had a fracture or sprain of either wrist so far as he could remember. He knew of no similar condition among his relations.

Radiological examination—In the left carpus the scaphoid was small and elliptical and lay transversely, its non-radial aspect being widely separated from the capitate bone and trapezium (Fig. 3). The general appearance of the scaphoid in this instance closely resembled that of the left side in Case 1. The right carpus was radiologically normal. *Cervical spine*—There

FIG. 4
Case 2—Cervical spine. Note spina bifida of the first thoracic vertebra.

was a spina bifida of the first thoracic vertebra (Fig. 4) and some degeneration of the cervical intervertebral discs, but no cervical ribs were present. *Intravenous pyelogram*—This showed crossed renal ectopia, both kidneys lying on the right side of the abdomen.

DISCUSSION

The two patients described were very like the patient in Hodgson's (1943) case in that they had a selective wasting of the thenar muscles in association with a carpal deformity which involved principally the scaphoid bone, and it seems probable that these lesions comprise a distinct entity. Unlike Hodgson's patient, neither of our patients had suffered much disability from the muscular lesions in spite of the almost total absence of opponens pollicis, a muscle that is of major importance in the power of the grip. It is difficult to be sure whether the affected muscles were wasted in the true sense or were in fact congenitally absent or maldeveloped. Both of our patients had lesions of the cervico-thoracic vertebrae which might conceivably have accounted for the muscular deficiency. Thus the patient in Case 1 had bilateral cervical ribs, and the combination of obliteration of the radial pulses with wasting of the thenar muscles might have suggested that the cervical ribs were responsible for both lesions. Nevertheless the complete absence of pain or sensory loss, and the presence of the muscular deficiency from early childhood, make it highly unlikely that the cervical ribs were in fact culpable. Similar arguments may be applied to the spina bifida in Case 2. All three

of the affected muscles of the thumb are supplied principally by the median nerve, and although it is possible that a lesion of a branch of the median nerve may have been the cause of the muscular lesions this is also unlikely in view of the total lack of sensory impairment.

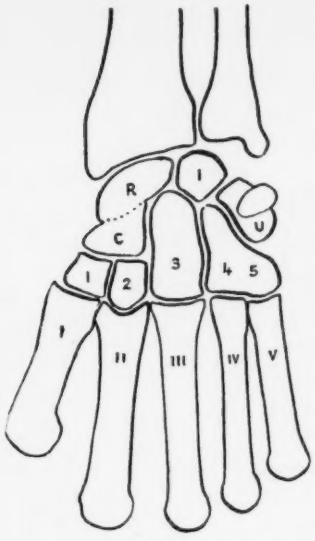

FIG. 5

Diagram of the origin of the normal human scaphoid according to Wood Jones (adapted from *The Principles of Anatomy as Seen in the Hand*, by F. Wood Jones, 1941).

Whatever the etiology of the muscular lesions there seems to be no doubt that the carpal deformity is of congenital origin. As with most congenital anomalies it is not possible to give a certain explanation of the origin of the lesions, but it is perhaps of interest that Wood Jones (1941) believed that " the human scaphoid is a compound bone, being composed of the primitive scaphoid in its proximal part and the pre-axially displaced os centrale in its distal part." In Figure 5 we reproduce Wood Jones's diagram, comparison of which with the radiographs of the left scaphoid bones in Cases 1 and 2 suggests that the deformity may possibly represent persistence only of the proximal part of the scaphoid, unfused with the distal part. Even if this were the correct explanation, however, it would not account for the other changes in the carpus, although the abnormalities of the first metacarpal might be related to the absence of its normal muscular attachments. It is difficult to envisage any direct relationship between the carpal deformity and the muscular deficiency, because none of the muscles involved has any material attachment to the scaphoid bone.

It seems probable that the condition described is less rare than the paucity of recorded cases would suggest, because it may cause relatively trivial symptoms and thus escape detection. In obscure cases of " wasting " of the intrinsic thumb muscles it may be worth while to radiograph the carpus.

We are grateful to Dr James W. Brown and to Professor C. H. Stuart-Harris for permission to publish reports of their patients.

REFERENCES

HODGSON, A. R. (1943): Congenital Retardation in Development of the Carpal Navicular, First Metacarpal, and Styloid Process of the Radius. British Journal of Surgery, **31**, 95.

JONES, F. Wood (1941): The Principles of Anatomy as Seen in the Hand. Second edition. London: Baillière, Tindall and Cox.

REDUCTION OF SMITH'S FRACTURE

F. BRIAN THOMAS, HEREFORD, ENGLAND

The Smith's or reversed Colles's fracture is a comparatively uncommon injury. The fracture and its treatment are described in the standard fracture text-books, but there appear to be no other references to it in the literature since Smith published his original paper describing the injury in 1847.

The fracture is easily recognised clinically (Fig. 1). The normal anterior curve of the lower end of the radius is exaggerated and the lower end of the ulna is abnormally prominent on the dorsum of the wrist.

FRACTURE OF THE LOWER EXTREMITY OF THE RADIUS, WITH DISPLACEMENT OF THE LOWER FRAGMENT FORWARDS.

This is an injury of exceedingly rare occurrence, and one which presents characters closely resembling those of dislocation of the carpus forwards. It generally occurs in consequence of a fall upon the back of the hand, and the situation of the fracture is from half an inch to an inch above the articulation; it is accompanied by great deformity, the principal features of which are a dorsal and a palmar tumour, and a striking projection of the head of the ulna at the posterior and inner part of the fore-arm; the dorsal tumour occupies the entire breadth of the fore-

arm, but is most conspicuous internally, where it is constituted by the lower extremity of the ulna displaced backwards; from this point, the inferior outline of the tumour passes obliquely upwards and outwards, corresponding in the latter direction to the lower end of the superior fragment of the radius. Immediately below the dorsal swelling there is a well-marked sulcus, deepest internally below the head of the ulna, directed nearly transversely, but ascending a little as it approaches the radial border of the fore-arm.

FIG. 1

Extract from Smith's original report of the fracture that bears his name.

No attempt appears to have been made to classify the different varieties of Smith's fracture. I have found it helpful to separate such fractures in adults into three distinct types which can be recognised radiologically.

Type 1—In the older patient, and particularly in women, the fracture extends across the lower cancellous end of the radius. The small lower fragment is bent forwards, and sometimes completely transposed anteriorly. It is often comminuted, and the ulnar styloid process is usually seen to be avulsed (Figs. 2 to 7).

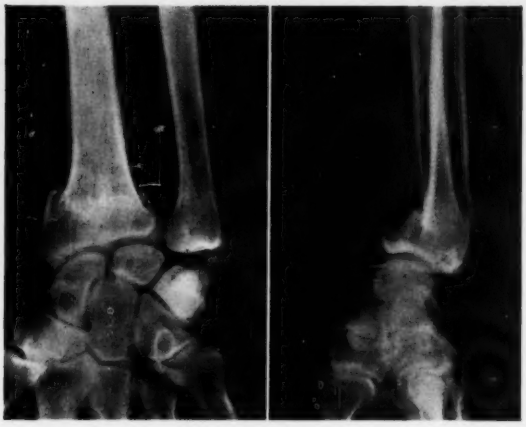

FIG. 2

FIG. 2
Case 1—Smith's fracture of Type 1. Before reduction.

FIG. 3

FIG. 3
Case 1—After reduction.

FIG. 4

FIG. 4
Case 1—After consolidation.

FIG. 5

FIG. 5
Case 2—Smith's fracture of Type 1. Before reduction.

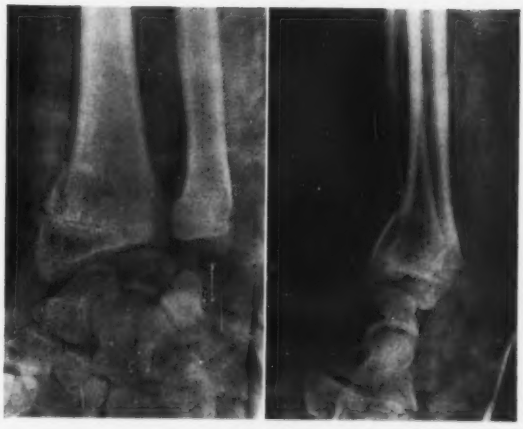

FIG. 6

FIG. 6
Case 2—After first attempt at reduction, with the forearm in neutral rotation.

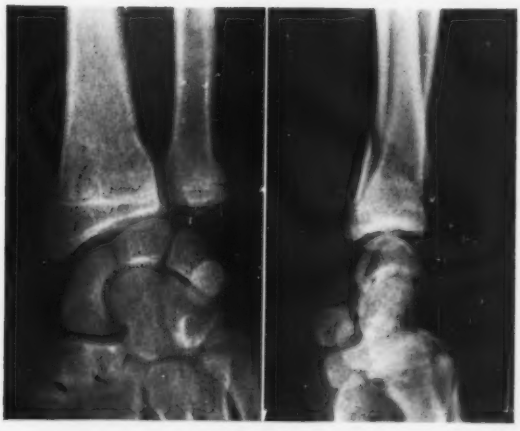

FIG. 7

FIG. 7
Case 2—After further reduction with full supination.

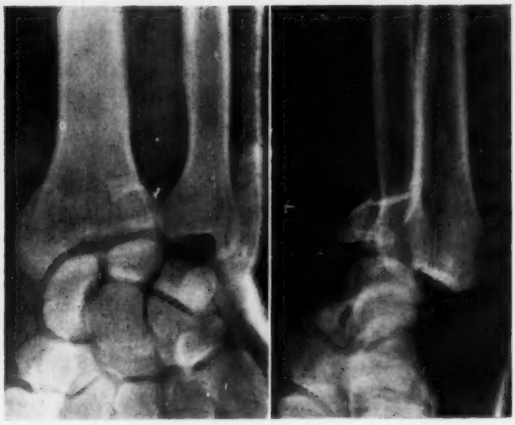

FIG. 8
Case 3—Smith's fracture of Type 2. Before reduction.

FIG. 8

FIG. 9
Case 3—After reduction.

FIG. 9

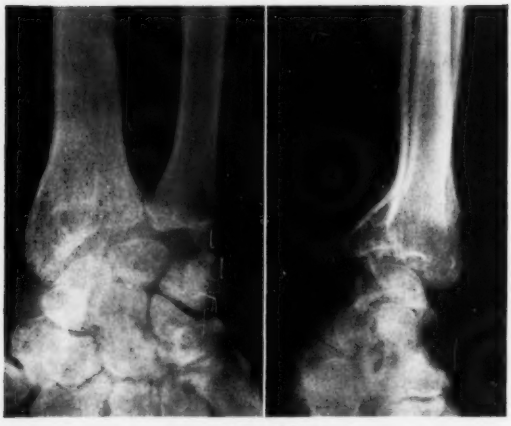

FIG. 10
Case 3—After consolidation.

FIG. 10

FIG. 11

Case 4—Smith's fracture of Type 3. Before reduction. Note the associated fracture of the second metacarpal.

FIG. 12

Case 4—After reduction.

FIG. 13

Case 4—After consolidation.

Type 2—In younger patients with harder bone, and particularly in men, the fracture takes a different form. There is an anterior marginal fracture of the lower articular end of the radius, with forward and proximal dislocation of the entire carpus (Figs. 8 to 10).

Type 3—The third type of Smith's fracture occurs almost exclusively in motor cyclists involved in accidents, and appears to be caused by a forcible blow on the knuckles of the hand as it grips the handlebar. The lower end of the radius shears within one inch of the wrist joint. The fracture line is seen to be roughly transverse in the antero-posterior radiograph. In the lateral view the fracture line is slightly oblique (Figs. 11 to 13). The lower end of the radius is displaced and tilted anteriorly. This type of fracture is frequently associated with fractures of the metacarpals.

In children the injury is seen as a forward fracture-separation of the lower radial epiphysis, or as a greenstick fracture of the radius within two inches of its lower end.

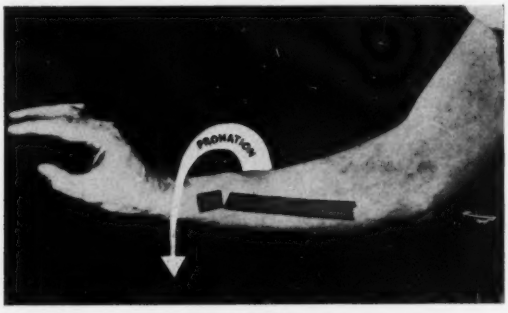

FIG. 14

A fracture sustained while the forearm is pronating will angulate backwards. (*Reproduced by permission of Mr Mervyn Evans.*)

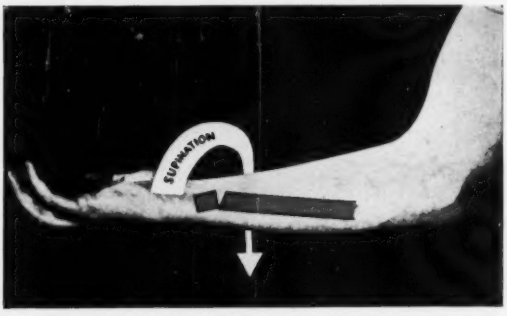

FIG. 15

A supination fracture will tend to angulate forwards. (*Reproduced by permission of Mr Mervyn Evans.*)

MECHANISM OF INJURY

Smith stated: " It (the fracture) generally occurs in consequence of a fall upon the back of the hand." His reference to the drawing (Fig. 1) accompanying his article has a strangely modern ring: " The patient, in endeavouring to save himself from being run over by a car, fell with great violence upon the back of the hand."

Few of the patients in my series were able to give an accurate account of the type of fall that caused the injury. Most of the fractures in men resulted from motor cycle accidents. None of the patients could recall falling upon the back of the hand, and no patient with

Type 1 or Type 2 fractures showed abrasions of the knuckles or dorsum of the hand, such as could have been expected to result from such a fall. Several patients, however, described a backward fall on to the palm of the outstretched hand.

In such a fall the forearm is locked in full supination, and the weight of the falling body pronates the upper limb with the hand relatively fixed to the ground. This twisting is combined with a heavy compressive force and, in theory at any rate, provides the ideal mechanism for the production of a Smith's fracture (Figs. 14 and 15) (Evans 1951).

Type 3 fractures, as mentioned before, are assumed to be caused by a severe blow on the knuckles of the clenched hand, and this assumption is confirmed by the occurrence of metacarpal fractures in this group.

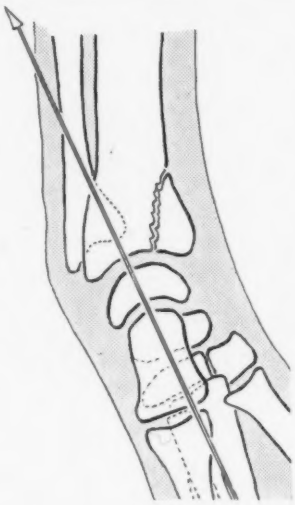

FIG. 16

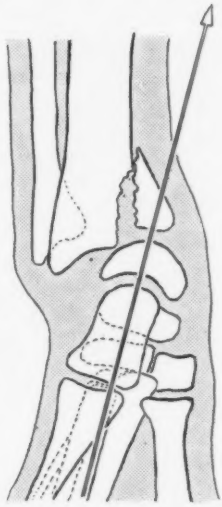

FIG. 17

The diagram in Figure 16 shows how the stability of a Smith's fracture of Type 2 is increased when the wrist is slightly flexed. Figure 17 shows how redisplacement of the fracture-dislocation may occur more easily when the wrist is extended.

TREATMENT

In all types of Smith's fracture reduction can usually be effected by a manipulation like that used for a Colles's fracture, but with all movements reversed. Maintenance of reduction is extremely uncertain, however, and redisplacement of the fracture almost invariably occurs, resulting in an ugly wrist with impaired function. Most orthopaedic surgeons seem to be aware of the poor results of treatment of this fracture, but none of the descriptions of treatment in the standard text-books of surgery stresses the difficulty of maintaining reduction by ordinary methods of splintage.

If it can be assumed that the Smith's fracture is caused by a pronation force, full supination of the hand would be expected to aid reduction. Maintenance of the fully supinated position should also prevent relapse of the fracture into a position of deformity.

In my series reduction was consistently achieved by forcing the hand into a position of extreme supination. The degree to which the hand should be supinated is determined by noting the greatest degree of supination possible in the opposite, uninjured limb. The fully supinated position was held by a plaster including the elbow, which was flexed at a right angle. Plaster fixation was continued for six weeks, and thereafter mobility was regained by normal active use of the limb.

Originally it was feared that fixation of these fractures in a position of full supination might be followed by some permanent restriction of pronation. With two exceptions, however, all patients in this series regained full pronation within three months.

One of the exceptions was an unusual fracture. The radial fracture was compound and there was a fracture of the shaft of the ulna which angulated while in plaster. The patient, a woman, had a permanent loss of pronation of 40 degrees. The other exception was a Type 2 Smith's fracture in a man who also sustained a severe head injury. This patient was reluctant to make active use of his injured wrist and hand, and he has an 80 degrees' loss of pronation.

The most unstable of all these fractures are those classified as Type 2. When this type of Smith's fracture is immobilised in a supination plaster the wrist should be slightly flexed. This position helps to maintain congruity between the proximal row of the carpus and the intact part of the lower end of the radius. Even a small degree of dorsiflexion encourages the carpus to subluxate forwards (Figs. 16 and 17).

CLINICAL MATERIAL

Smith's fractures are uncommon, and in the past three years I have managed to collect only nineteen cases. It is not intended to make a detailed analysis of the results of treatment; apart from the two patients mentioned above, all regained normal function.

Some of the patients were seen several days after the primary "casualty" reduction. With one exception the fractures had all either become redisplaced after an initially acceptable reduction, or had never been satisfactorily reduced. The first fracture in this series (a Type 2 fracture) was two weeks old when finally reduced by manipulation into full supination. The classification of the nineteen fractures of this series is shown in Table I.

TABLE I

NINETEEN SMITH'S FRACTURES CLASSIFIED ACCORDING TO TYPE

Type of fracture	Number of cases			Remarks
	Male	Female	Total	
Type 1 . . .	1	8	9	
Type 2 . . .	3	1	4	
Type 3 . . .	2	—	2	Both patients had associated metacarpal fractures
Fractures in children			4	One fracture-separation of lower radial epiphysis. Three fractures of the shaft of the radius within two inches of the wrist joint

SUMMARY

A classification of Smith's fractures into three types is proposed. It is suggested that the majority of these fractures are caused by a pronation injury. The reduction of Smith's fractures by supination is described and the importance of the supinated position during splintage is stressed.

REFERENCES

EVANS, E. M. (1951): Fractures of the Radius and Ulna. Journal of Bone and Joint Surgery, **33–B,** 548.

SMITH, R. W. (1847): A Treatise on Fractures in the Vicinity of Joints, and on Certain Forms of Accidental and Congenital Dislocations, pp. 129–175. Dublin: Hodges and Smith.

WATSON-JONES, Sir R. (1943): Fractures and Joint Injuries. Third edition. Vol. 2, pp. 549–551. Edinburgh: E. & S. Livingstone Ltd.

UNREDUCED ANTERIOR DISLOCATION OF THE HIP

GAWAD HAMADA, ALEXANDRIA, EGYPT

Anterior dislocation of the hip is uncommon compared with posterior dislocation. Thompson and Epstein (1951) collected 204 cases of dislocation of the hip from the Los Angeles Hospital records in twenty-one years. Of these, 180 cases were posterior whereas only eighteen were anterior.

In the last ten years seven cases of anterior dislocation presented themselves to us. Three dislocations were acute and were reduced on admission. The remaining four were old unreduced dislocations.

Dislocations seen early are easily reduced by the well known manipulative methods. Old unreduced dislocations present much greater problems. A number of cases of unreduced posterior dislocation have been reported, but unreduced anterior dislocation is almost unheard of. Searching extensively in the literature the author has not come across any recorded case like those to be reported in this paper.

MECHANISM OF INJURY

Traumatic dislocation—Two forces may be responsible for the production of anterior dislocation. One is a force from the knee through the femur while the limb is in a position of extreme flexion, abduction and lateral rotation. This type of injury is exemplified by one of our patients who fell while riding astride on a donkey, her knee striking the ground (Case 1). The second type of injury is a force applied to the inner side of the thigh, forcing it into extreme lateral rotation and abduction, the limb being in a position of flexion. This type of injury was graphically related by one of our patients who was admitted with an acute anterior dislocation. He gave a history that he was supporting the weight of his body on his right flexed limb while the left limb was resting on a higher level in the position of flexion. He was piling sandbags, when suddenly the mound slipped and hit the inner part of his flexed thigh, felling him to the ground and resulting in an anterior dislocation.

Pathological dislocation—This may be caused by infantile paralysis, tumours or inflammatory conditions. The dislocation is produced through the prolonged maintenance of a position that facilitates slipping of the head of the femur anteriorly. This is shown in Case 2.

CLINICAL FEATURES

The patient presents an exaggerated lumbar lordosis with scoliosis towards the affected side compensating the hip deformity. The pelvis is lower on the affected side. The Trendelenburg test is positive in the old chronic cases. The limb is abducted, laterally rotated and slightly flexed at the hip and the knee.

In the pubic type of dislocation the head may be felt lying over the pubic bone under cover of the origin of the adductors. In the obturator type the head is not felt. The greater trochanter is difficult to palpate because it is pushed nearer to the midline and because the limb is markedly abducted.

Movements of the hip are generally lost, with the exception of flexion and abduction which are greatly reduced. Real lengthening is slight, but apparent lengthening is marked. The gait is laboured and the strides are short and jerky, the patient putting little weight on the affected side. In walking, the patient fixes the extended thigh by placing the hand over the knee, in the manner of a patient with quadriceps paralysis. Since the femoral head is displaced outside the acetabular socket, the abductor muscles become tense and cause flexion

FIG. 1

FIG. 2

Case 1—Patient before and after treatment.

FIG. 3

FIG. 4

Case 1—Radiographs before and after corrective osteotomy.

of the hip. The pelvis rolls to the front; thus the Trendelenburg lurch in these cases occurs more towards the front than to the side. This explains why the patient places his hand on his thigh, to prevent further rolling forward of the pelvis, and thus attempts to stabilise his gait.

TREATMENT

The aim of any operative intervention is to correct the deformity and improve body mechanics and balance. An osteotomy is done at the base of the neck, from above downwards and medially. The limb is then slightly adducted, extended and medially rotated. The abductor muscles attached to the greater trochanter sometimes pull the shaft slightly upwards on the base of the neck, rendering the upper end of the femur L-shaped. This will give better stability to the pelvis, thus distributing the weight of the trunk on the affected side between the trochanter and the head, and not solely on the head (see Case 3). After operation the limb is placed in a plaster spica which includes the sound limb to the knee. Union is usually firm in three to four months.

FIG. 5

Case 2—Attitude maintained by the patient during the active stage of the arthritis: abduction of the affected hip and adduction of the sound hip.

CASE REPORTS

Case 1—Woman aged twenty-four years. This patient had fallen from a donkey twelve years before. Radiographs (Fig. 3) showed the femoral head, small and atrophied, lying opposite the obturator foramen. The acetabulum and the iliac bone were undeveloped, probably because the injury had occurred at an early age, before union of the epiphysis. The femoral head was probably affected by avascular necrosis in consequence of the dislocation. Operation was done and resulted in the improvement of the deformity, stability and gait (Figs. 1 to 4).

Case 2—Girl aged sixteen years. The history was of pain in the left hip with rise of temperature. An abscess was incised. The patient remained in the sitting position for two years; the affected hip was abducted, laterally rotated, and touching the bed. The right hip was adducted and lay over the left thigh. Two years after the onset, when the inflammation had subsided, she gradually started to extend her limbs and when she stood up she found that she had developed the deformity. Osteotomy was done with improvement of the condition (Figs. 5 to 9).

FIG. 6

FIG. 7

Case 2—Photographs of the patient before and after corrective osteotomy.

FIG. 8

FIG. 9

Case 2—Radiographs before and after corrective osteotomy.

FIG. 10
Case 3—Radiograph before operation.

FIG. 11
Case 3—After corrective osteotomy.

Case 3—Boy aged twelve years, who had fallen from a height a year before his admission to hospital. The deformity was the same as in the previous cases. Osteotomy was done and the deformity corrected (Figs. 10 and 11).

Case 4—Girl aged nine years. She had slipped and rolled down a flight of stairs five months before admission. Corrective osteotomy was done with satisfactory results (Fig. 12).

FIG. 12
Case 4—Radiograph after corrective osteotomy.

SUMMARY

1. Unreduced anterior dislocation of the hip is very uncommon. Four cases are reported.
2. The aim of treatment is to correct the deformity, to improve the gait and to produce pelvic stability.
3. Osteotomy of the femur at the trochanteric level is the simplest corrective operation and will greatly improve the function of the limb.

REFERENCE

THOMPSON, V. P., and EPSTEIN, H. C. (1951): Traumatic Dislocation of the Hip. Journal of Bone and Joint Surgery, **33-A**, 746.

ARTHRODESIS OF THE HIP WITH A LAG-SCREW

G. K. McKee, Norwich, England

A method of arthrodesis of the hip by the use of a lag-screw which acts by combined compression and fixation has been used since 1948, and the first fifty cases reviewed two years or more after operation show that bony fusion was obtained in 94 per cent, and in 76 per cent of the cases the use of plaster was avoided.

The apparatus—The apparatus consists of a cylindrical shaft of stainless steel, seven-sixteenths of an inch in diameter, with a coarse corkscrew-like thread at one end for gripping the ilium and a more finely threaded lower end for attachment to a three-hole plate (Fig. 1). The apparatus is made with two lengths of shaft, one four inches and the other three and a half inches.

The coarsely threaded upper end of the apparatus is screwed into the acetabulum and obtains a firm grip on the bone of the ilium. The three-hole plate is shaped to fit the convexity of the shaft of the femur, and the square hole for attachment to the lower end of the lag-screw is bevelled on its upper edges so that it will easily ride over the threads when the connecting nut is tightened. At the operation the shaft is made a sliding fit in the drilled-out neck of the femur, and after attachment of the three-hole plate, tightening of the nut holding the two together will cause compression as well as firm fixation of the joint.

Special instruments required—Two box spanners are used, one for holding and inserting the lag-screw and one for tightening the connecting nut.

A series of ordinary twist drills are used; these are mounted on shafts to make them up to a length of ten inches. The sizes are graduated from three-sixteenths inch to seven-sixteenths inch, the last one being of the same diameter as the shaft of the lag-screw.

It is advisable to have a low-geared hand drill for use with the larger drills, because they are too coarse to cut compact bone easily in the ordinary hand drill.

TECHNIQUE OF OPERATION

Position—The patient suitably anaesthetised is placed on an orthopaedic table with the limb in the chosen position for arthrodesis. Shortening may be compensated for by abduction, and old patients should have a few more degrees of flexion than the younger ones. The limb should also be in a few degrees of lateral rotation. The position is carefully marked so that after it has been altered during the operation the limb may be returned to this previously selected position. It is useful to tilt the table laterally if possible, because the operation is carried out mainly from the lateral aspect.

Incision and approach—The incision extends from the anterior superior iliac spine downwards and backwards to the greater trochanter and then curves more directly downwards in the line of the upper end of the shaft of the femur (Fig. 2).

The gluteal fascia is incised in the line of the wound and over the greater trochanter, and the approach is as far as possible between the tensor fasciae latae muscle in front and the gluteal muscles behind, exposing the capsule of the hip joint. The gluteal muscles are stripped off the deeper part of the wing of the ilium and the greater trochanter is removed; this improves the exposure and provides a useful block of bone for an ilio-femoral graft later.

If the incision through the gluteal fascia in the lower part of the wound is extended it exposes the vastus lateralis muscle, and at a later stage this muscle is reflected from the side of the upper end of the shaft of the femur to expose the area of bone for the insertion of the lag-screw and the application of the three-hole plate.

The intra-articular procedure—After the excision of the joint capsule the cartilage is removed from the head of the femur and from the acetabulum by a large curved gouge (Fig. 3). In cases of osteoarthritis the head is often considerably enlarged, and it is necessary to remove enough bone to allow impaction of the head in the acetabulum. To do this effectively, an

FIG. 1

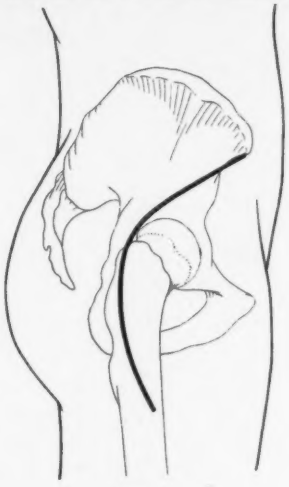

FIG. 2

Figure 1—The lag-screw and plate. There are two lengths of shaft—3½ inches and 4 inches.
Figure 2—Incision for lateral approach.

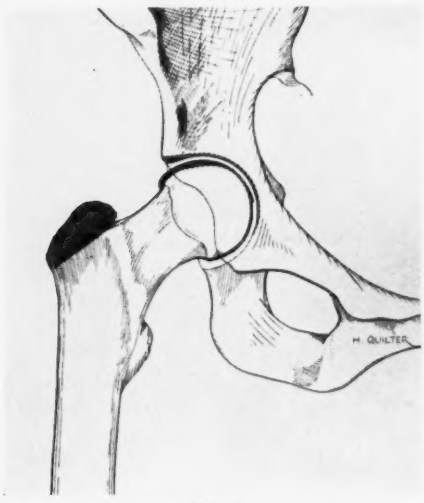

FIG. 3

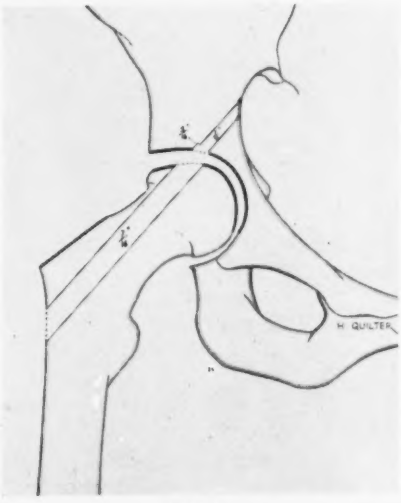

FIG. 4

Figure 3—The greater trochanter, and the articular cartilage of the acetabulum and of the head of the femur, are removed. Figure 4—Preliminary drilling before the insertion of the screw. The neck of femur has been drilled to $\frac{7}{16}$ inch and the ilium to $\frac{1}{2}$ inch.

assistant manipulates the limb into various positions of abduction, adduction and rotation so that all aspects of the head come into view. The head is not actually dislocated because tension is kept on the limb, but it is fairly widely rotated out of the acetabulum. Light traction on the leg opens up the joint space and is helpful in improving the access to the interior of

the joint. When sufficient cartilage and bone have been removed, the traction is released to make certain that the raw surfaces will come together. The full and free apposition of raw bony surfaces is essential for compression to be effective, and it ensures rapid bony fusion.

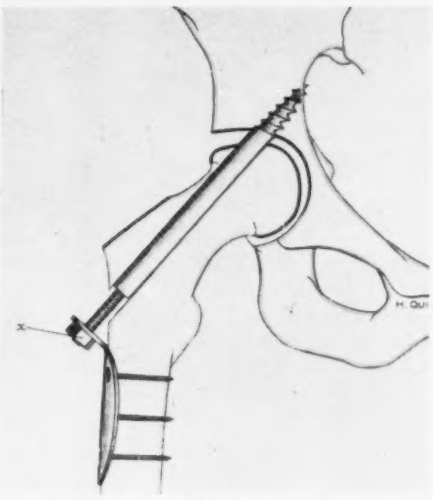

FIG. 5

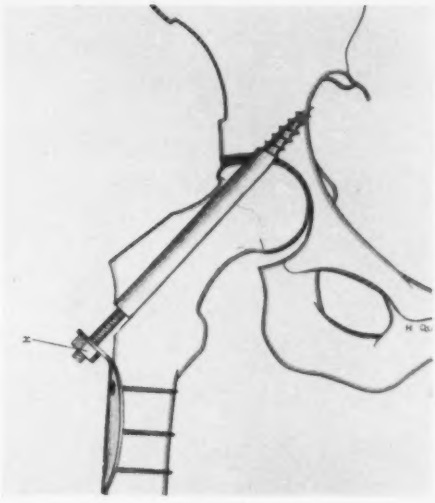

FIG. 6

Figure 5—The lag-screw and plate have been inserted. The shaft of the screw has a sliding fit in the neck of the femur; so compression of the joint surfaces can be obtained by tightening the main nut (*x*). Figure 6—The upper part of the joint space has been obliterated by tightening the main nut (*x*).

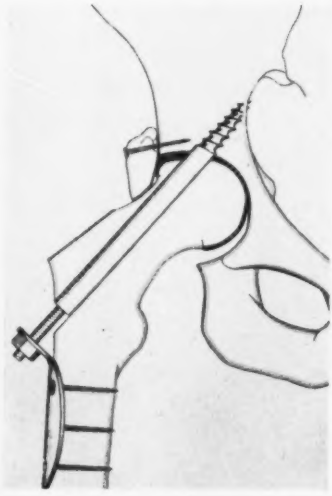

FIG. 7

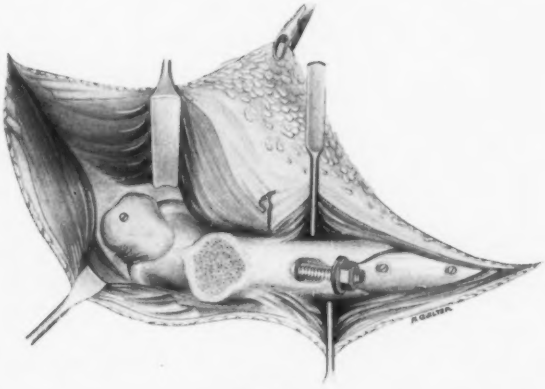

FIG. 8

Figure 7—The arthrodesis completed, as seen from the lateral aspect. Figure 8—The completed arthrodesis shown diagrammatically. The greater trochanter bridges the gap between the head of the femur and the acetabulum and is fixed in place by a screw.

Insertion of the lag-screw—The position of the limb is checked and adjusted if necessary to the selected position for fusion, and a slight pull is put on the leg to open up the joint space so that later, when the track of the lag-screw is drilled out, the position of the drill can be seen as it crosses the joint.

A point is selected on the lateral aspect of the upper end of the femoral shaft about an inch and a quarter below the base of the greater trochanter, and the cortex of the femur at this point is thinned over an oval area by a gouge. This facilitates the subsequent drilling.

A three-sixteenths inch drill is directed through the neck of the femur until it is seen to impinge on the acetabular roof towards the lateral aspect. The direction of the drill should be as vertical as possible without coming too close to the upper surface of the neck of the femur.

The length of drill shaft projecting when the drill point engages the acetabulum and when it encounters the inner cortex of the ilium are noted, and thus the length of bone traversed in the ilium can be estimated. This should be at least an inch and a quarter. It is essential to drill right through the inner cortex, which can be felt by its increased resistance to the drilling. At this stage great care must be taken not to go through the cortex with an uncontrollable jerk.

FIG. 9
Radiograph showing apparatus inserted and firm bony ankylosis of the hip.

The pilot hole is enlarged by successively wider drills. The quarter inch drill is driven well into the ilium, but the five-sixteenths inch drill is penetrated only as far as the acetabulum, and no more. The three-eighths inch and the seven-sixteenths inch drills are used only in the femoral neck and head, so that the shaft of the lag-screw will be a sliding fit (Fig. 4).

A lag-screw of a suitable length is selected; it is fairly simple to measure the length required but it is important to avoid changing the lag-screw once it has been inserted, otherwise a certain amount of looseness of grip may result. The slight pull on the limb should be released while the screw is being inserted and the shaft should be seen to cross the joint line, thereby ensuring that the strongest part of the apparatus is where the greatest strain occurs. After the apparatus has been firmly screwed home the joint space should have closed up.

A small three-hole plate is now attached by a plain nut to the end of the lag-screw and by three screws to the lateral aspect of the femoral cortex (Fig. 5). When this plate has been attached to the femur further tightening of the nut which attaches the plate to the lower end of the lag-screw will force the upper end of the femur tightly against the acetabulum. Blood clot and debris are usually squeezed out of the joint when this nut is tightened (Fig. 6).

Very firm impaction should be obtained, so that when the limb is rocked no movement is seen to occur at the line of arthrodesis.

Additional grafts—Additional extra-articular grafts are probably not necessary, but, as the greater trochanter is so readily available, it is usually replaced across the joint line as an ilio-femoral graft and fixed in position by a screw holding it to the ilium. Cancellous bone fragments may also be taken from the cut surface from which the greater trochanter was removed, and placed about the joint line (Figs. 7 to 9).

In some of the earlier cases an iliac crest graft was also used, but this does not seem to be necessary, and since it requires additional exposure and adds to the length of the operation it has been given up.

Post-operative care—When secure fixation was obtained at the operation (76 per cent of cases) the patient was allowed up on crutches without plaster during the third week, and gave up the crutches for sticks before the sixth week. In some cases the patients were back at work in six weeks and could walk well without sticks before the twelfth week.

If there was any doubt about the hold of the screw in the ilium a short hip spica was applied two weeks after the operation, before the patient began getting up. The plaster was usually kept on for six weeks.

ANALYSIS OF THE FIRST FIFTY CASES

The first fifty consecutive cases of disease of the hip (other than tuberculosis) in which this method of arthrodesis was performed were assessed personally by the author both before and at least two years after operation.

Selection of cases—The selection of cases for operation has an important influence on the results, and it is usual to keep the patients under observation for a time—sometimes for a year

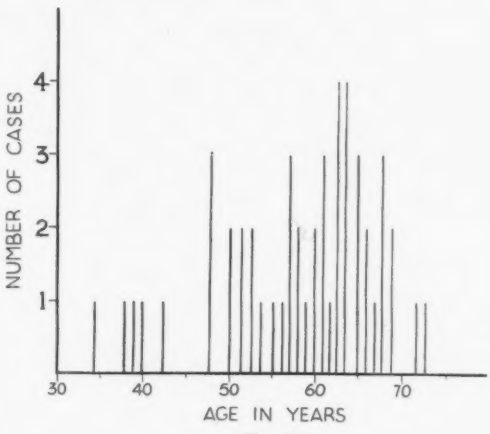

FIG. 10
Age distribution.

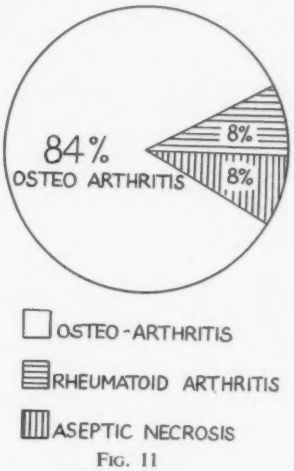

FIG. 11
Diagnosis.

or two—before the operation is carried out. A period of observation of this nature confirms that osteoarthritis of the hip is a progressive disease, and most patients gradually become worse if conservative measures alone are employed.

The idea of a stiff hip is often a stumbling block to the lay mind, but if the patient hesitates it is a good plan to arrange for him to see a patient who has already had the operation; this never fails to convince him of the advantages of having it done.

Arthrodesis is the method of choice in severe arthritis of the hip, especially if the disease is unilateral and in a young patient, but in older patients arthroplasty may be preferred. Arthrodesis is also useful in bilateral disease; it should be carried out on the more severely affected side to give a strong weight-bearing limb on that side. The other hip, relieved to some extent from strain, may never require operation, but if necessary an arthroplasty may be performed later.

The radiographic appearance is also helpful in deciding on the advisability of operation, as it is a valuable guide to the degree of arthritis present.

Age distribution—The average of the patients was fifty-eight years. The graph of age distribution (Fig. 10) shows a concentration of patients between forty-seven and sixty-nine years.

Sex—Twenty of the first twenty-five patients were women, sixteen of the second twenty-five were men. Twenty-nine of the whole series were women.

Diagnosis (Fig. 11)—The condition requiring treatment by arthrodesis was osteoarthritis in forty-four cases, rheumatoid arthritis in four and ischaemic necrosis of the head of the femur in four. The cases that gave difficulty and delay in union were the cases of ischaemic necrosis, and if operation had been reserved for cases of arthritis only, the rate of fusion would have been considerably higher.

Post-operative assessment—Arthrodesis of the hip is in many ways simpler to assess than arthroplasty, and in contrast to that procedure it seems that the longer the interval between operation and the final assessment, the better the result. Three factors—fusion, pain and function—have been considered in the assessment and will be dealt with in detail later.

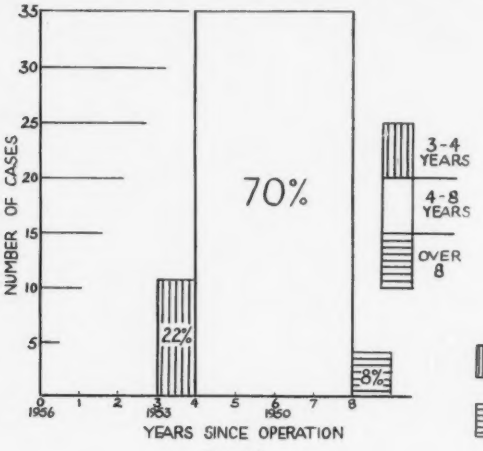

FIG. 12
Interval between operation and assessment.

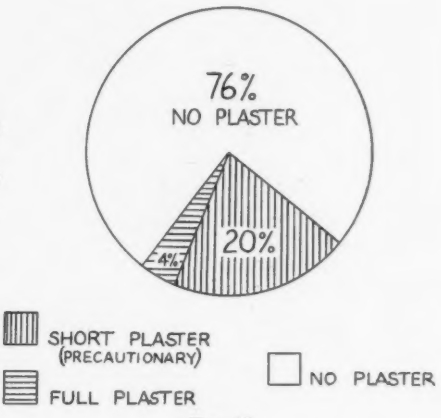

FIG. 13
The avoidance of plaster.

Interval between operation and assessment (Fig. 12)—Every patient in this series had the operation performed over two years before the final assessment, and in all the more recent cases bony fusion occurred in the first year. Most patients (70 per cent) have been observed for four to eight years.

Avoidance of plaster—The avoidance of the use of plaster fixation is one of the advantages claimed for this method of arthrodesis, and it was possible to do without it entirely in thirty-eight of the fifty cases (Fig. 13). In ten cases a short plaster hip spica was applied, usually two weeks after the operation, before the patient began getting up. Two patients required a full-length hip spica, one on account of sepsis (Case 8) and the other because the internal fixation was insecure (Case 16). Both eventually obtained firm bony fusion after considerable delay.

Fate of the lag-screw—The screw remained intact in thirty-four of the fifty patients. The screw broke in eight cases, but in only half of these did it need to be removed, because the other patients had obtained bony fusion and were free from symptoms. Breakage of the screw seemed most liable to occur in cases of ischaemic necrosis, and in cases in which it was not screwed home far enough, so that the threaded part crossed the joint line instead of being embedded in the ilium. In eight cases the screw was removed intact on account of minor symptoms, mainly tenderness on pressure over the lateral aspect of the upper part of the thigh.

Knee movement—The average range of knee movement was 95 degrees (Table I). Thirty-five of the fifty patients had a range of movement from 180 degrees to 90 degrees or more after the operation. There was not a complete record of the range before the operation in all the cases, but the two patients with the poor range and the two with incomplete extension had the same range of knee movement before as after operation. The only cases in which there was known to be a decrease in the knee movement after operation were the two in which a long plaster hip spica had to be applied.

TABLE I
RANGE OF KNEE MOVEMENT AFTER OPERATION (FORTY-SEVEN CASES)

Grading of result	Range of knee movement	Number of cases
Good	180–90 degrees or more	35
Fair	180–140 degrees	8
Poor	180–160 degrees	2
Incomplete extension	160–70 degrees 170–70 degrees	2

The two patients graded as " poor " and the two with incomplete extension had impaired movement before the operation.

The preservation of a full range of knee movement is important to a patient who has a stiff hip because it enables him to put his shoes and socks on without the help of gadgets or other people. The avoidance of plaster is an important factor in allowing more rapid and complete restoration of knee movement.

RESULTS

An estimation of the results of this method of treatment has been obtained by combining the three main and important features of fusion, pain and function. Three marks were allowed for each, so that a maximum of nine marks represents an excellent result. In practice this was found to be a simple and accurate method of assessment when compared with the general clinical picture of the patient's condition.

Bony fusion—Bony fusion was estimated from clinical and radiological evidence. Forty-seven of the fifty patients obtained bony fusion eventually (Fig. 14). If the patients that died are disregarded, there remains only one case in which bony fusion failed to occur; that was a case of massive aseptic necrosis (Case 11).

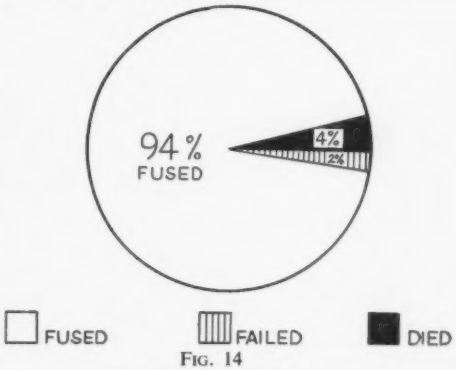

FIG. 14
Incidence of bony fusion.

There is obviously a definite relationship between pain and fusion, although some of the patients in whom fusion was undoubtedly sound did experience slight pain at times in the hip, but not on movement or weight bearing; so the question of pain is assessed separately.

Trabecular continuity—Trabecular continuity is radiological proof of fusion, but it always takes many months to appear. There was not a complete series of radiographs showing trabecular continuity in all the cases, but in the twenty-four that were available it was found that the time taken for the appearance of trabecular continuity in the early cases was over two years; in the later cases, after better compression was obtained, the time was reduced to one year (Table II). These investigations are incomplete, but the findings suggest that

the measures taken to ensure better compression in the later cases brought about the more rapid appearance of trabecular continuity.

Pain—The absence of pain is easy to record, but the degree of pain is difficult to establish because it varies so with individuals. At the time of the final assessment, pain in the hip,

TABLE II

ESTABLISHMENT OF TRABECULAR CONTINUITY

	Number of cases with trabecular continuity	Time for establishment of trabecular continuity
Whole series of 50 cases	24	1·6 years
First 25 cases . .	10	2·4 years
Second 25 cases . .	14	1 year

groin, buttock or thigh was absent in thirty-four of the fifty patients and slight in twelve (Fig. 15). "Slight pain" consisted in the main of aching in the hip at various times. A moderate degree of pain was present in one patient. The patient in whom fusion failed to occur is classed as unrelieved, and two deaths account for the remainder.

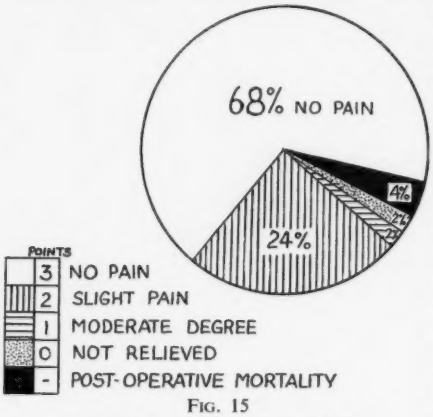

FIG. 15

Incidence of pain after operation.

Function—The position in which fusion occurs is the most important factor in determining the functional result. There is a definite optimum position for each individual patient, and it is important to obtain this position to ensure a good result. It is important to avoid excessive abduction because, with a fixed hip, it is easier to walk with about half an inch of apparent shortening. A satisfactory functional result was obtained in forty-three patients, graded excellent and good (Fig. 16). The four patients in the "fair" grading showed some degree of adduction deformity which was responsible for the impairment. The failure was in the case in which bony fusion was not obtained.

Combined assessment of fusion, pain and function—Figure 17 shows the results given by a combined estimation of the three salient features—fusion, pain and function. Seventeen patients gained excellent results, sixteen good, and fourteen fair. The standards in this estimation are higher than when one feature alone is considered, and "fair" represents a satisfactory result—one in which patient and surgeon are quite pleased with the success of the operation—and this or better was obtained in forty-seven of the fifty patients.

Aseptic necrosis of the femoral head—Aseptic necrosis of the femoral head is still a major problem in orthopaedic surgery. There were four cases of this condition dealt with in this series. In three there was massive necrosis of the head after fracture of the neck of the femur; in the fourth case there was localised necrosis after a trochanteric fracture. In the patient with localised necrosis (Case 46) fusion occurred very slowly; he wore a protective plaster for over a year, but eventually obtained a good result (eight points).

In the three patients with massive necrosis fusion was even more difficult and slow. In one patient (Case 11) there was complete failure to secure fusion—the only total failure in the whole of this series. The lag-screw broke and was removed seven months after the arthrodesis operation, and a Whitman's reconstruction and cup arthroplasty was carried

out. Although the arthrodesis was classed as a complete failure, this patient can walk without a stick and is fairly satisfactory four years after the arthroplasty. Of the remaining two patients one (Case 7) gained bony fusion very slowly, the screw broke and adduction deformity occurred. The screw was removed and a subtrochanteric osteotomy was performed, and eventually fusion took place but with one and three-quarters inches of shortening. The other patient (Case 8) was also very slow to gain fusion, and the case was further complicated by a

GRADING	MARKS	NUMBER OF CASES	%
EXCELLENT WALK LONG DISTANCES NO AIDS CAPABLE OF MANUAL WORK AS FARMERS, BLACKSMITHS: FULL H/D.	3	20	40
GOOD WALK LIMITED DISTANCE CAN SIT DOWN AND STAND UP BY THEMSELVES.	2	23	46
FAIR REQUIRE STICK TO WALK WITH	1	4	8
FAILED	0	1	2
DIED		2	4

FIG. 16
Function at last assessment.

low-grade wound infection. The screw was removed and a plaster applied. Fusion eventually took place in some adduction and the patient required a raised shoe but had no pain. She walked well, could sit down and stand up by herself, and her own comment was that she was " much better than before the operation." This was classed as a fair result.

Aseptic necrosis unsuitable for this type of arthrodesis—Although three out of four of these cases of aseptic necrosis eventually gave satisfactory results, fusion was delayed and they

GRADING	MARKS	NO. OF CASES
EXCELLENT	9	17
GOOD	8	16
FAIR	7	14
FAILURE	0	1
DIED	-	2

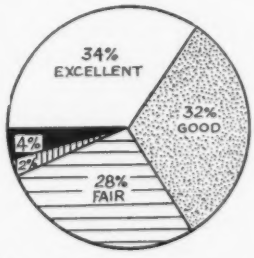

FIG. 17
The combined assessment of fusion, relief of pain and function. Three marks are allowed for each. Nine marks are therefore the maximum for any one case and represent an excellent result.

were the most difficult of all the cases to deal with. In my opinion arthrodesis of the hip, or at any rate this type of arthrodesis, is not suitable for cases of aseptic necrosis of the femoral head.

COMPLICATIONS

Fracture of the femur—Fracture of the shaft of the femur seems to be one of the hazards of a successful arthrodesis of the hip. Two main factors are responsible for this: firstly, the patients become very active and take liberties, and yet have an increased liability to fall; and secondly, the strain imposed on the shaft of the femur by a fall is increased by the fixity of the hip.

Fractures of the femur occurred in four cases, all in women. One was only a crack without displacement, between the site of insertion of the lag-screw and the plate. The other

three were complete fractures at or just below the level of the plate. All occurred in active patients who had definite falls. All these patients did well eventually.

Backache—Backache was not a conspicuous symptom in any of the patients, although most of those with osteoarthritis admitted to backache before the operation. The backache persisted after the operation but was never a prominent complaint even five or six years afterwards.

Wound infection—There was no case of severe wound infection, but a low-grade infection occurred in two patients. One of these had massive necrosis of the femoral head which took some months to clear up; the other had only a superficial infection which subsided in a few weeks.

Mortality—There were two deaths in this series after the primary operation, one from post-operative shock thirty-six hours after operation (Case 13), and one from coronary thrombosis three weeks later (Case 50).

A further patient (Case 1) died after a secondary operation six years after the arthrodesis. She complained of tenderness over the apparatus and it was decided to remove the lag-screw and plate. This proved difficult because the screws holding the plate were so firm that they could not be unscrewed and had to be chiselled out of the bone, with the result that a fracture of the femur occurred and a plaster hip spica was applied. Nine days later the patient died suddenly from a pulmonary embolism. The moral to be learned from this is to refrain from attempting to remove the apparatus for minor symptoms, especially if it has been in a long time.

MODIFICATIONS OF TECHNIQUE

There were a few " teething troubles " in the early stages, such as the tendency of the lag-screw to break in cases of ischaemic necrosis. Such cases are not now considered suitable for this type of arthrodesis, and since they have been eliminated and since a stouter lag-screw has been used, in no case has the screw broken in the last fourteen cases of this series and in a further twenty-four that have been carried out since. It is important to make certain that the shaft of the screw—not just the threaded part—is seen to cross the joint space, and to obtain firm compression of the bony surfaces; both these details are important in lessening the strain on the screw and in promoting rapid bony union.

SUMMARY AND CONCLUSIONS

A method of intra-articular arthrodesis of the hip is described in which fixation is obtained with a lag-screw. Two main advantages are claimed: first, that it is a compression arthrodesis and gives early bony fusion; and second, that it gives adequate internal fixation in most cases so that the disadvantages of a plaster hip spica are avoided, and the patient may walk with crutches two or three weeks after the operation.

The results were satisfactory in forty-seven of the first fifty cases in which this method of arthrodesis was employed. There were two post-operative deaths and one failure to secure fusion in a case of massive necrosis of the femoral head.

FLEXION-COMPRESSION INJURY OF THE STERNUM

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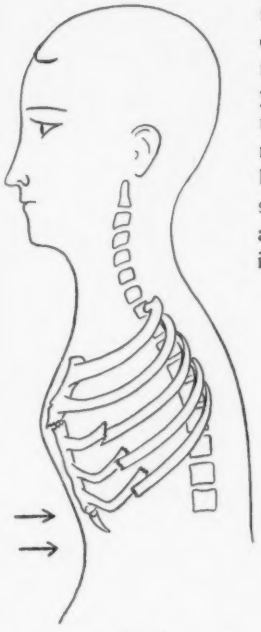
Injuries of the sternum are commonly due to either direct violence to the front of the chest or indirect violence from flexion-compression of the spine. Injuries from hyperextension are exceedingly rare and will not be considered further. Nowadays many injuries of the sternum result from road accidents. Crushing injuries are frequently due to the impact of the steering column, and flexion-compression injuries may occur if the vehicle turns over or if passengers are flung out. In order to emphasise the distinctive features of the flexion-compression injury a short description of both kinds of sternal injury will be given.



Direct injuries—Direct violence may cause two different types of sternal injury. The first and by far the commoner is due to crushing of the lower part of the front of the chest. The lower mobile part of the sternum receives the impact, but the sternum yields at a variable level above the site of impact. The sternum usually gives way near the junction of the mobile body with the relatively fixed manubrium in the same way that a branch of a tree breaks off near the trunk (Figs. 1 to 3). The second type of direct sternal injury occurs when the blow on the chest is localised and at a higher level (Figs. 4 and 5). The sternum is injured at the site of impact, and one fragment of the sternum may be displaced behind

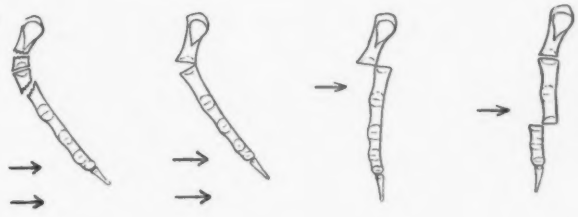


FIG. 1 FIG. 2 FIG. 3 FIG. 4 FIG. 5

Injuries from direct violence. Figures 1 to 3—Crushing of lower part of sternum causing angulation above site of impact. Figures 4 and 5—Blow at higher level causing injury at site of impact.

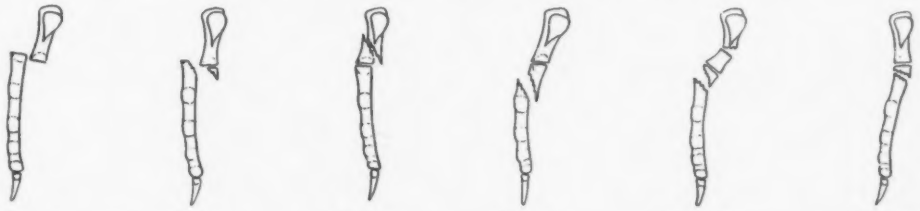


FIG. 6 FIG. 7 FIG. 8 FIG. 9 FIG. 10 FIG. 11

Flexion-compression injuries. The sternum gives way in or near the manubrio-sternal joint. The injury may cause a dislocation (Fig. 6), fracture-dislocation (Fig. 7), oblique fracture (Figs. 8 and 9), or fracture with backward angulation (Figs. 10 and 11).

FIG. 12

Case 1—Tomograph showing fracture of upper part of body of sternum with backward angulation and compression fracture of T.4.

FIG. 13 FIG. 14 FIG. 15

Case 2. Figure 13—Posterior dislocation of munubrium. Figure 14—Compression fracture of T.9. Figure 15—Sternum after open reduction and wiring.

the other. The commonest variety of this injury is a posterior dislocation of the body of the sternum on the manubrium.

Direct sternal injuries are often accompanied by rib fractures (Fig. 1), and in severe cases there is paradoxical movement in the anterior chest wall. The skin is bruised and the underlying viscera may be damaged. Local symptoms therefore are marked and the pain, which is aggravated by breathing, causes considerable distress.

Flexion-compression injuries—In contrast to the two types of direct injury, flexion-compression violence produces one distinctive type of sternal injury. This injury has two characteristic features: 1) it is always in the first two segments of the sternum—usually at or near the manubrio-sternal joint; 2) the upper or manubrial fragment is depressed in relation to the lower fragment. The commonest injury is a dislocation or fracture-dislocation of the manubrio-sternal joint with backward and downward displacement of the manubrium on the body of the sternum (Figs. 6 and 7). In other cases flexion-compression causes a fracture of either the manubrium or the upper part of the body, with a similar displacement of the upper on the lower fragment (Figs. 8 and 9). Rarely the injury takes the form of a fracture of the manubrium or upper part of the body with backward angulation (Figs. 10 and 11).

Flexion-compression injuries are frequently associated with head injuries and fracture of the spine (Figs. 24 and 25). Pain in the chest is relatively mild, and the clinical picture is often dominated by the more serious injuries to the head and spine. The clinical differences may be summarised by saying that flexion-compression injury of the sternum may be present but not suspected, whereas direct injury may be suspected even when it is not present. Six cases of flexion-compression injury of the sternum will be described.

CASE REPORTS

Case 1—A woman aged twenty-nine was the front seat passenger in a car which rolled down a hillside. She complained of severe pain in the chest and back, but radiographs of the ribs showed no bone injury. Treatment was given for a dislocation of the left elbow and a cut over the left temple. Two months later the pain in the back persisted and a swelling over the sternal angle was noted. Radiographs of the sternum were then taken, and showed a fracture just below the manubrio-sternal joint and a compression fracture of T.4 vertebral body (Fig. 12). Treatment consisted of breathing and postural exercises. The pain in the sternum gradually subsided but she was left with some permanent tendency to backache.

Case 2—A man aged twenty was flung from the pillion seat of a motor cycle which ran into a ditch. He felt an immediate constriction in the chest and was unable to speak for a few minutes. When seen later he was not distressed but complained of pain in the front and back of the chest. There was a step deformity at the sternal angle but no bruising or evidence of a blow on the sternum. Radiographs showed a posterior dislocation of the manubrium and small compression fractures of T.8 and 9 vertebral bodies (Figs. 13 and 14). The dislocation was not reduced by extension of the spine and on the third day open reduction was undertaken. The manubrio-sternal joint was exposed through a transverse incision over the sternal angle. The second ribs were found to be attached to the manubrium. After soft tissues had been cleared from between the fragments the manubrium was pulled forward by means of a curved spike inserted into a drill hole. The reduction was unstable, and suture of the soft tissues was inadequate to hold it securely. A Kirschner wire was drilled obliquely into the body of the sternum and up into the manubrium. The distal end of the wire was allowed to protrude an inch, and it lay almost parallel with the skin (Fig. 15). The patient reported immediate relief from discomfort in the chest. He was discharged on the sixth day and after three weeks the wire became loose and came out. He was forbidden to return to heavy work for a further month. After a further month he was able to resume his occupation as a coal miner. When seen three months after the injury he had no symptoms and a radiograph showed a normal manubrio-sternal joint.

Case 3—A farmer aged fifty-nine rolled off a hay wagon and landed on the back of his neck. He complained of pain at the base of the neck and tingling in both arms. He had abrasions over the occiput and left scapula. There was a depression of the upper part of the sternum which disappeared when he lay down but recurred on sitting up. There was no bruising or abrasion on the front of the chest and he did not complain of pain in the sternum. Radiographs showed an oblique fracture of the second sternal segment with depression of the manubrium and a fracture-dislocation of the

FIG. 16

FIG. 17

FIG. 18

Case 3. Figure 16—Oblique fracture of sternum with depression of manubrium. Figure 17—Fracture-dislocation of C.5. Figure 18—Sternum after wiring under radiographic control.

fifth cervical vertebra (Figs. 16 and 17). The patient was laid flat on an operating table and a Kirschner wire was inserted through a skin puncture and drilled across the sternal fracture under radiographic control (Fig. 18). A plaster jacket was applied, with a headpiece holding the neck extended. After three weeks the wire was withdrawn and after three months the plaster was removed. Both the sternum and the cervical spine healed without deformity.

FIG. 19

Case 4—Fracture of manubrium after reduction.

Case 4—A man aged thirty-five fell fifty feet on to his head. He sustained severe concussion, an extensive laceration of the scalp, a fracture of the left clavicle, and an oblique fracture of the manubrium. There was no evidence of a blow on the sternum. Figure 8 is drawn from a poor quality radiograph taken on admission, when there was a marked depression of the manubrium. This deformity disappeared with a click when the spine was extended over a pillow placed behind the scapulae (Fig. 19). Radiographs of the spine showed wedging of the upper thoracic vertebrae but no definite evidence of a recent fracture. The patient was kept in bed for three weeks because of his head injury. The sternal deformity did not recur and required no further treatment.

Case 5—A man aged sixty-two was descending a ladder while facing away from it. The lower end of the ladder slipped, and he dropped eight feet and landed on his buttocks. He complained of pain in the back and over the upper part of the sternum. On examination there was tenderness over the whole spine, and especially in the cervico-thoracic and thoraco-lumbar regions. There was a slight swelling at the sternal angle, and when he coughed the manubrium appeared to move backwards. Radiographs showed a fracture of the manubrium with slight backward displacement of the upper fragment (Fig. 20), and a small compression fracture of the first lumbar vertebra (Fig. 21). After a short period of recumbency he was allowed up. His only complaint from the sternum was a pain on coughing.

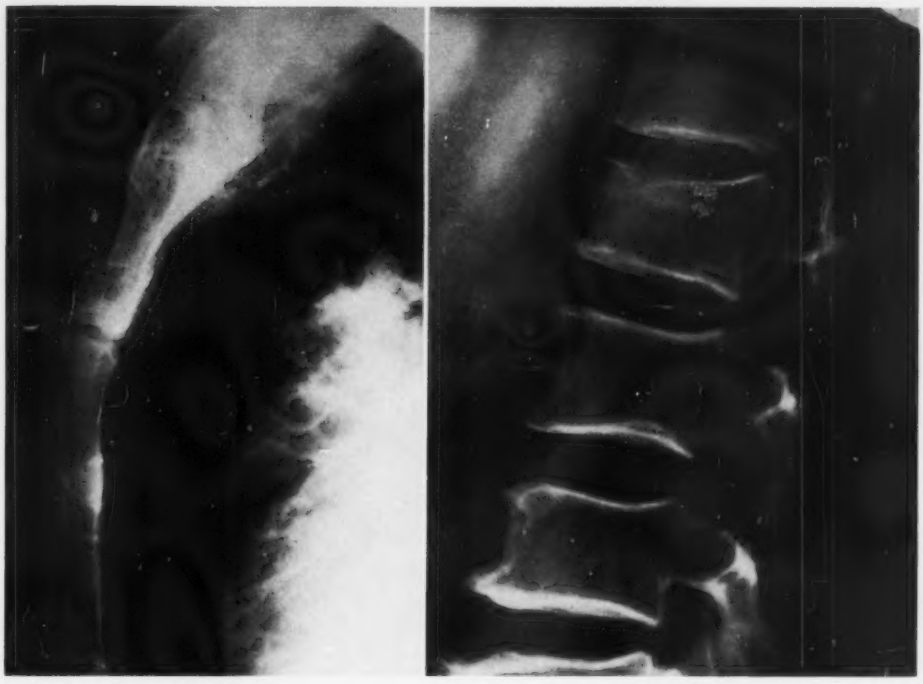


FIG. 20 FIG. 21

Case 5. Figure 20—Fracture of manubrium. Figure 21—Compression fracture of L.1.

Case 6—A miner aged forty-five was in a sitting position when a heavy stone fell two feet on to the back of his head. He was not concussed and had a clear recollection of the accident. His head was forced down but his chin did not strike forcibly against his chest. There was a small cut over the occiput and a fracture through the second piece of the sternum with slight backward displacement of the upper fragment; there was no gross injury to the cervical spine.

TREATMENT

Reduction of the displacement in manubrio-sternal flexion-compression injuries is usually simple: laying the patient flat and extending the thoracic spine over a pillow or the end of a table is usually effective. When the sternal injury is stable after reduction and the spinal injury is insignificant no further treatment is necessary (Cases 4 and 5). But if the sternal deformity recurs when the patient sits up it is necessary to maintain extension either by recumbency or by splintage. The usual treatment is by a frame or a plaster jacket; an alternative is to use some form of internal splint. The experience with Case 2 shows that a Kirschner wire gives adequate fixation and allows the patient complete freedom of movement. The wire was redundant in Case 3 but it demonstrated how easily a wire can be inserted under radiographic control, using a local anaesthetic if necessary.

If there is an important spinal fracture, as in Case 3, that should take precedence in treatment. Reduction of the spinal displacement will usually correct the sternal deformity and the same fixation will maintain the reduction of both injuries.

Occasionally manipulation fails to reduce a posterior dislocation of the manubrium and operation is necessary to secure reduction (Case 2). Two similar cases have been described before. Hartzell (1935) after elevating the manubrium held it in place with periosteal sutures. The subsequent development of dense callus and a sequestrum at the site of operation suggests

TABLE I

FIFTEEN FLEXION-COMPRESSION INJURIES OF THE STERNUM DESCRIBED DURING THE LAST FIFTY YEARS

Author and date	Sex	Age	Nature of accident	Nature of sternal injury	Associated injuries
Alexander 1906	M	60	Fell 8 feet on to head	Fracture of sternum just above level of third ribs; lower fragment slipped forwards and upwards on upper fragment	Bruise and cut on back of head above the occiput
Brunn 1913	M	49	Hit on neck by 1 cwt. sack of hides	Fracture of manubrio-sternal junction with transverse ridge palpable	Painful mid-thoracic kyphosis
	M	53	Hit on neck by 3 cwt. object	Manubrium displaced posteriorly with step deformity	Painful mid-thoracic kyphosis
	F	45	Hit on neck by 80 lb. sack of tobacco leaves	Manubrium completely dislocated posteriorly and overlapping body by 1 centimetre	Painful mid-thoracic kyphosis. Fracture of fibula
Pazzi 1926	—	—	Thrown from car, striking occipital region	Anterior dislocation of body on manubrium	—
Holderman 1928	M	20	Squeezed between trams in a colliery	Anterior dislocation of upper end of body at the joint	Compression fracture of L.2
Pendergrass 1929	F	55	Motor accident	Posterior depression of manubrium on body; fracture of manubrium and upper end of body	Compression fracture of T.4
Rothbart 1929	F	42	Fell from window on to head	Fracture of manubrium with dorsal displacement	Contusion of occipital region
Brown 1931	—	—	" Jacknifed " under car with head forced between knees	Complete posterior dislocation of manubrium and fracture of upper end of body	—
Stuck 1933	M	44	Crumpled forward by cave-in of ditch	Fracture of manubrium	—
	F	75	Motor accident	Fracture of manubrium	Compression fractures of T.4 and 5
Ellis 1935	—	—	Powerful forward flexion from fall on buttocks	Irregular oblique fracture of upper end of body running into joint, with slight forward displacement of lower fragment	—
Hartzell 1935	M	32	Thrown through top of car	Posterior dislocation of manubrium	Contusions of occipital region
Kirkham 1941	F	67	Fell downstairs with head flexed forwards as in gambolling	Fracture of sternum at junction between manubrium and body. Manubrium displaced behind body	Compression fracture of T.5
Robertson 1955	M	13	Severe tetanus	Depressed fracture of manubrium	Compression fractures of T.4 to 8

that this fixation was inadequate. McKim (1943) fixed the fragments with two Kirschner wires, one driven into the manubrium and the other into the right second rib. The subcutaneous situation of the sternum makes open reduction and wire fixation such a simple procedure that it is recommended in all irreducible dislocations of the sternum.

DISCUSSION

This study of flexion-compression injuries of the sternum is based on six personal cases and fifteen cases described during the last fifty years (Table I). Table I includes only those injuries in which flexion-compression can be inferred with certainty either from the history of the accident or from associated injuries.

TABLE II

ANALYSIS OF ACCIDENTS CAUSING FLEXION-COMPRESSION INJURY OF THE STERNUM

	Fall of patient on head or back	Weight falling on head or neck	Crushing violence	Fall of patient on buttocks	Road accidents without details of how injury was caused	Muscular contractions
21 cases (1906–1956) collected by author	6	5	3	2	4	1
17 cases (1853–1905) collected by Tarnowsky	12	2	2	1	—	—
Totals	18	7	5	3	4	1

Etiology—This is analysed in Table II and compared with seventeen cases from the previous half-century collected by Tarnowsky (1905). About a third of the injuries are due to road accidents. Changes in methods of transport are reflected in the fact that in all the road accident cases in Tarnowsky's series the patients had fallen off their vehicles, whereas to-day the injury occurs in those who have been flung out of vehicles or rolled about inside them. A detailed history of the accident is often lacking because the frequent association of concussion makes an accurate reconstruction of the accident impossible. I have been careful to exclude from this study any sternal injuries in which there is evidence that direct violence was applied to the chest. The sternal injury due to tetanus (Robertson 1955) is included in this series because it was clearly caused by flexion-compression of the thoracic spine which in turn was caused by powerful muscular contractions.

The sternal injury—A comparison of Figures 6 to 11 will show that all the flexion-compression injuries of the sternum are basically similar. The sternum gives way at or near the manubrio-sternal joint. The anatomy of the joint determines whether the injury takes the form of a dislocation, fracture-dislocation or fracture. Dislocations are likely to occur only when the manubrio-sternal joint is synovial. Rivington (1874) examined 100 manubrio-sternal joints and found the following types: cartilaginous fifty-one, synovial thirty-two, intermediate eleven, and ossified six. He found the synovial type of joint more frequently in adults than in children and postulated that it was formed by absorption of cartilage during growth. A recent study by Ashley (1954) has shown that synostosis of the manubrio-sternal joint is not a senile change but is found in about 10 per cent of all subjects after the age of thirty. These anatomical studies explain why dislocation of the manubrio-sternal joint usually occurs in adults and may occur in the elderly.

In dislocation of the manubrio-sternal joint the second rib always remains attached to the manubrium. This is due to the fact that when the manubrio-sternal joint is synovial the cartilage of the second rib is much more firmly attached to the manubrium than to the body of the sternum (Maisonneuve 1842). Lane (1884) explained that the movements of the body of the sternum upon the relatively fixed manubrium caused absorption of cartilage and the formation of synovial joints between the body on the one hand and the manubrium and second rib on the other.

There is no general agreement about the exact nature of the displacement in this injury. Eight authors described the injury as a posterior displacement of the manubrium and four described it as an anterior displacement of the body of the sternum (Table I). I believe that in most cases the manubrium is displaced backwards. The radiological appearances in Case 1, the operation findings in Case 2 and the clinical features in Case 3 indicate that the manubrium was depressed in these patients. The nature of the displacement will depend on the mechanism of the injury, and in the discussion on mechanisms I shall give reasons for believing that the upper fragment is depressed in at least two-thirds of these injuries.

The spinal injury—Nine out of twenty-one flexion-compression injuries in this series were accompanied by proven fractures of the spine, comprising six fractures of the thoracic spine, one fracture of the cervical spine and two fractures of the lumbar spine. The fracture of the spine in Case 2 was only detected by careful radiography; it is therefore possible that some patients in Table I had undetected fractures of the thoracic spine. In four road accident cases the spinal fractures provided the only clue to the mechanism of the sternal injury.

The spinal injuries are usually simple wedge fractures. This indicates a large vertical component in the force applied to the spine (Fig. 22). Comminuted fractures and fracture-dislocations of the spine are caused by acute angulating forces (Fig. 23) and are not accompanied by fractures of the sternum. It is for this reason that fractures of the sternum do not complicate

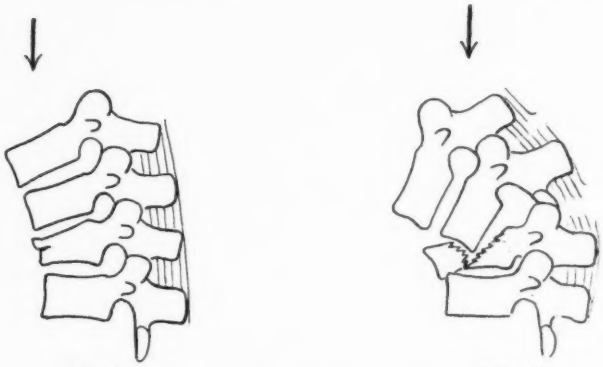


FIG. 22 FIG. 23

Figure 22—Simple wedge fractures are caused by forces acting mainly along the spine. Figure 23—Comminuted fractures and fracture-dislocations are caused by forces acting on a bent spine.

the thoraco-lumbar fractures and fracture-dislocations in miners, which are due to a fall of roof on a bent back. The sternal injury from tetanus provides us with a "slow motion" study of the accompanying spinal injury. Robertson (1955) stated that the spasms caused vertical compression of the spine, the head being drawn down vertically towards the thoracic inlet.

Mechanisms of the sternal injury—There are three possible routes by which the forces might be transmitted to the sternum: 1) along the clavicles, 2) by the descending chin, and 3) along the ribs. Maisonneuve (1842) and Lane (1884) attached importance to the part played by the clavicles. This is difficult to understand in view of the mobility of the clavicles and the fact that they lie mainly in the coronal plane. Martin (1901) recorded a fracture of the manubrium caused by a lateral impact on the clavicle; this caused a vertical fracture of the left edge of the manubrium with forward displacement. Lane (1884) produced an oblique fracture between the second costal cartilages without displacement by dropping a weight vertically on the outer end of the clavicle in a cadaver. Neither of these injuries corresponds to the typical flexion-compression injury of the sternum; both were caused by blows over the outer end of the clavicle, whereas flexion-compression injuries are caused by forces acting along the spine. The only circumstances in which the clavicles might be brought into line with such forces are in falls on the fully extended and outstretched arms. Tuttle (1885) reported a manubrio-sternal dislocation in a man who dived into shallow water. He postulated that the force was transmitted to the manubrium along the outstretched arms and through the clavicles. His argument is weakened by the fact that his patient said that he fell on his head. Moreover no other flexion-compression injury of the sternum has been recorded in which the victim landed on his outstretched arms.

With regard to the descending chin, I agree with the conclusion of Rivington (1874) that it plays a part mostly, if not entirely, in cases of injury to the cervical spine (Fig. 25). It is easy to demonstrate this in the cadaver; even when the manubrio-sternal ligaments have been divided the chin will cause dislocation of the manubrium only after the cervical spine has been fractured. If the chin were responsible for displacing the manubrium one would expect to find bruising beneath the jaw; no evidence of this is found in any of the recorded cases.

It is probable that the ribs play the most important part in the transmission of forces to the sternum. Convincing evidence of this can be obtained in the cadaver. The sternum is removed by dividing the costal cartilages close to the bone, and movements of the rib ends

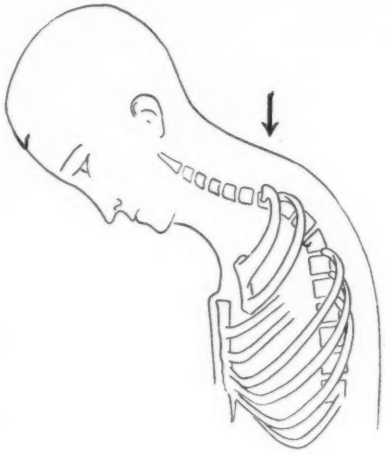


FIG. 24

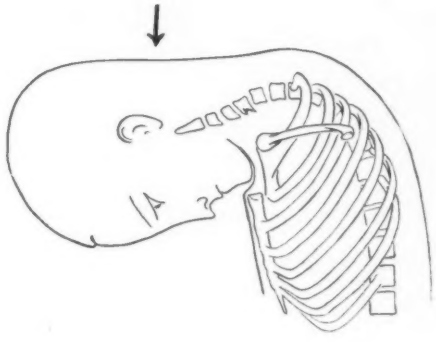


FIG. 25

Mechanisms of flexion-compression injury. Figure 24—Fall or blow on upper part of spine. Flexion-compression fracture of thoracic spine. Manubrium twisted off body by upper ribs. Figure 25—Blow or fall on back of head and neck causing fracture of cervical spine. Descending chin may assist in depressing manubrium.

are observed while the spine in manipulated. Flexion of the neck and thoracic spine imparts a downward and backward movement to the upper ribs—particularly the upper two ribs. Flexion of the lumbar and thoraco-lumbar spine has no effect on the ribs until the posterior abdominal wall abuts against the thoracic cage—the lower ribs are then pushed upwards and forwards. Therefore in a uniform flexion of the spine the sternum is subject to two opposing forces: the manubrium is forced downwards and backwards by the upper two ribs and the body of the sternum is forced upwards and forwards by the lower ribs. The sternum yields at its weakest point between these two forces.

I believe that in the majority of flexion-compression injuries the upper ribs dominate in the production of the sternal deformity, the manubrial fragment being twisted off the rest of the sternum. In eleven out of twenty-one cases the injury was known to be caused by a blow or fall on the head or neck, and six out of nine proven fractures of the spine were above the mid-thoracic level. In both of these groups, which together comprise two-thirds of the cases, we can infer that the upper part of the spine was flexed on the lower and that the manubrial fragment was displaced by the upper ribs (Fig. 24). There is a group of five cases due to crushing injuries and falls on the buttocks in which it may be assumed that the lower part of the spine was flexed and that the sternal deformity may have been partly or wholly due to the upthrust of the lower ribs.

There are anatomical reasons for believing that the upper two ribs must take an active part in displacing the manubrium. The manubrium is firmly attached to the spine by short and relatively rigid ribs which effectively transmit a twisting force from the spine to the manubrium. The body of the sternum, however, is joined by longer and more flexible ribs which will only transmit a flexion force from the spine indirectly. Several authors (Servier 1889, Tarnowsky 1905, Stuck 1933) argued that the manubrium, being solidly fixed to the spine, retains its position, whereas the body of the sternum, articulating with longer and more mobile ribs, is displaced. This would be true if flexion-compression were confined to the lower part of the spine. But when the upper spine is flexed the manubrium being firmly attached to the spine will be moved out of position.

The first costal cartilages are massive and hold the upper two-thirds of the manubrium in a vice-like grip. It seems likely therefore that the first rib is chiefly responsible for the control of the manubrium. When the manubrium itself is fractured the line of fracture is at the junction of the upper two-thirds with the lowest third (Figs. 8 and 10); the level of this fracture is further evidence of the importance of the first rib in the mechanism of the injury.

SUMMARY

1. Injuries of the sternum from flexion-compression violence are described and contrasted with injuries from direct violence. The characteristic feature of flexion-compression injuries is a dislocation or fracture at or near the manubrio-sternal joint with backward displacement of the manubrium.

2. Treatment is outlined, with special reference to the indications for open reduction and wire fixation.

3. The mechanism of flexion-compression injuries of the sternum is discussed. The opinion is expressed that most of the force is transmitted to the sternum by the ribs—especially the upper ribs.

I wish to thank Mr G. Hyman, Mr J. G. H. James, Mr H. J. Richards, Mr G. Rowley and Mr J. N. Wilson for allowing me to treat their cases. I am grateful to Mr J. G. H. James and Mr H. J. Richards for helpful criticism.

REFERENCES

ALEXANDER, J. F. (1906): A Case of Fracture of the Sternum. Lancet, **ii,** 936.
ASHLEY, G. T. (1954): The Morphological and Pathological Significance of Synostosis at the Manubrio-sternal Joint. Thorax, **9,** 159.
BROWN, R. (1931): Dislocation of Manubrium of Sternum. Surgical Clinics of North America, **11,** 933.
BRUNN, W. v. (1913): Über Frakturen des Sternum. Zentralblatt für Chirurgie, **40,** 633.
ELLIS, J. D. (1935): Fractures of the Sternum. Surgical Clinics of North America, **15,** 605.
HARTZELL, J. B. (1935): Fracture of the Sternum. Annals of Surgery, **102,** 158.
HOLDERMAN, H. H. (1928): Fracture and Dislocation of the Sternum. Annals of Surgery, **88,** 252.
KIRKHAM, J. H. (1941): Depressed Fracture of the Sternum. British Medical Journal, **ii,** 614.
LANE, W. A. (1884): One Mode of Fracture of the Sternum. Transactions of the Pathological Society of London, **35,** 253.
MCKIM, L. H. (1943): A Method of Fixation for Fractures of the Sternum. Annals of Surgery, **118,** 158.
MAISONNEUVE, J. G. (1842): Recherches sur la luxation des deux premières pièces du sternum. Archives Générales de Médecine, **3,** série **14,** 249.
MARTIN, J. S. (1901): A Case of Fracture of the Sternum. Lancet, **i,** 617.
PAZZI, E. (1926): Lussazione anteriore del corpo dello sterno sul manubrio messa in evidenza con la indagine radiologica. Archivio di Radiologia, **2,** 569.
PENDERGRASS, R. C. (1929): The Roentgenographic Demonstration of Sternal Injury, with Report of a Case. Radiology, **13,** 451.
RIVINGTON, W. (1874): Remarks on Dislocations of the First and Second Pieces of the Sternum. Medico-Chirurgical Transactions, **57,** 101.

ROBERTSON, D. H. H. (1955): Kyphosis and Fracture of the Manubrium in Tetanus. Journal of Bone and Joint Surgery, **37–B,** 466.
ROTHBART, L. (1929): Fratura manubrii sterni. Fortschritte auf dem Gebiete auf dem der Röntgenstrahlen, **40,** 89.
SERVIER (1889): Mécanisme des luxations du sternum. Gazette Hebdomadaire de Médecine et de Chirurgie, **2,** série **26,** 154.
STUCK, W. G. (1933): Fractures of the Sternum. American Journal of Surgery, **N.S. 22,** 266.
TARNOWSKY, G. de (1905): Contrecoup Fracture of the Sternum. Annals of Surgery, **41,** 252.
TUTTLE, J. P. (1885): Coincident Fracture and Dislocation of the Sternum by Indirect Force. Medical Record, **28,** 681.

ANEURYSM COMPLICATING TRAUMATIC SPONDYLOLISTHESIS

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A man aged twenty-six years was admitted to the Royal Victoria Infirmary two hours after a coal mining accident. He had been struck on the back and right side by a large mass of falling stonework. Immediately after the injury he had had pain in the back and in the left leg, but on admission the pain in the leg had been replaced by severe left-sided abdominal pain.

On examination the patient was pale and sweating; the blood pressure was 85/50 millimetres Hg. and the pulse rate 96 per minute. Multiple abrasions over the back indicated

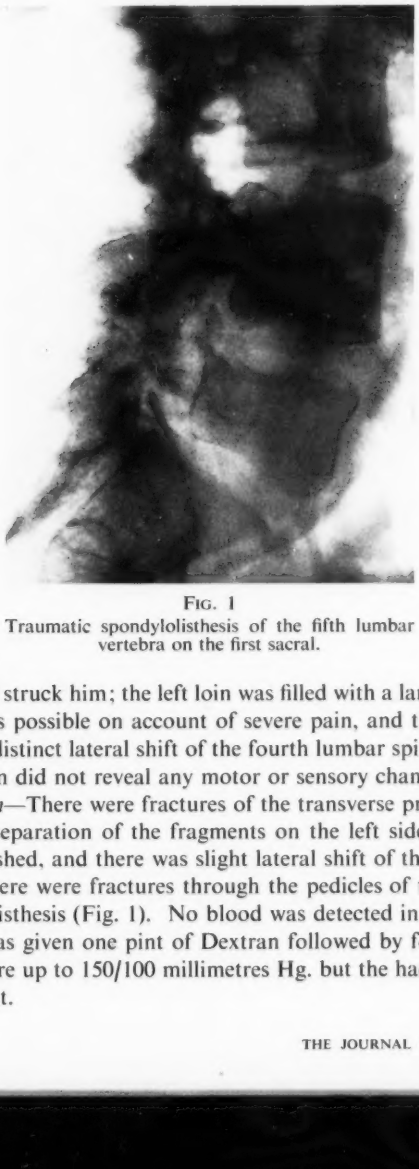

FIG. 1
Traumatic spondylolisthesis of the fifth lumbar vertebra on the first sacral.

where the stonework had struck him; the left loin was filled with a large fluctuant haematoma. No spinal movement was possible on account of severe pain, and there was tenderness over the lumbar spine with a distinct lateral shift of the fourth lumbar spinous process on the fifth. Neurological examination did not reveal any motor or sensory change in the limbs.

Radiographic examination—There were fractures of the transverse processes of all the lumbar vertebrae with extreme separation of the fragments on the left side. The body of the fifth lumbar vertebra was crushed, and there was slight lateral shift of the fourth lumbar vertebra on the fifth (Fig. 2). There were fractures through the pedicles of the fifth lumbar vertebra, with moderate spondylolisthesis (Fig. 1). No blood was detected in the urine.

Progress—The patient was given one pint of Dextran followed by four pints of blood. This brought his blood pressure up to 150/100 millimetres Hg. but the haemoglobin level two days later was only 60 per cent.

During the next week the patient's condition gradually improved, and it was decided to immobilise the spine in a plaster bed for three months. At this time the swelling in the left loin had diminished and become soft, only a small bulge being visible.

In the second week the plaster bed was made but on his return to the ward after this the patient complained of severe pain in his back and down the medial aspect of the left thigh from the groin to the knee. On examination he was distressed and pale; the lumbar swelling was again tense and tender and had returned to its former dimensions. The blood pressure was 140/90 millimetres Hg. and the pulse rate 100 per minute. Over the next three days the haematoma gradually became less tense and the pain subsided; at the end of this time the haemoglobin was 78 per cent.

In the third week the patient was placed into the plaster bed. At first he tolerated the immobilisation well, but after three days, while being turned to have the pressure areas treated, he complained of severe pain in the back. The following day he was taken out of the plaster bed as the tension of the haematoma made it impossible for him to remain in it with comfort.

By the sixth week the patient's condition was good, the haemoglobin being 92 per cent and the haematoma soft. It was decided to place him once more in the plaster bed, but the next day he had to be removed because he was complaining of pain in the back and of numbness and pain in the left thigh. This numbness persisted and evacuation of the haematoma was considered advisable.

First operation (seventh week)—A small oblique incision was made over the most prominent part of the haematoma in the loin. A large cavity was entered and from this one and a half pints of blood clot were removed. The wound was resutured without drainage and healed by first intention.

During the eighth week, while having physiotherapy to the legs, the patient complained of sudden severe pain in the back and weakness of the left leg. He also noticed that the haematoma had become larger and harder. On examination there were weakness of the quadriceps and sensory impairment in the distribution of the femoral nerve. Over the next two days the femoral nerve lesion became complete and the haematoma appeared even larger. The haemoglobin dropped to 76 per cent; platelet counts, clotting and bleeding times were within normal limits. Radiographs of the lumbar spine showed no increase in the vertebral displacement, but the fractured lumbar transverse processes on the left showed greatly increased separation (Fig. 2).

At this stage aortography was carried out because an aneurysm leaking intermittently was considered to be a real possibility. This failed to reveal any abnormality of the abdominal aorta or of the iliac arteries; the lumbar arteries however were not displayed. An excretion pyelograph performed at the same time is reproduced in Figure 2; it shows well the state of affairs, the whole loin being filled with haematoma grossly displacing the fractured transverse processes outwards and lifting the ureter forwards and inwards.

By now it was evident that the patient's life was being endangered by recurrent massive haemorrhage, for he looked pale and ill and was loosing weight steadily; the haemoglobin was 72 per cent and eight pints of blood had been given over the course of his illness. It was decided to explore the haematoma with full preparations to repair or remove an aneurysm of one of the main vessels.

Second operation (ninth week)—The haematoma was entered through a loin incision. A great mass of clot was disclosed and gently ladled out; in all five pints of soft clot were evacuated, and the walls of the cavity were composed of a further layer of organising thrombus over half an inch thick. During this process a firm, rounded swelling, adherent to its surroundings, was felt close to the lower lumbar spine; this was left undisturbed until the cavity had been completely cleared. Careful inspection showed that the swelling was about two inches across, and it could be seen and felt to pulsate vigorously. At this stage it was impossible to be certain from which vessel the aneurysm arose, and adequate exposure of the abdominal aorta and

left iliac vessels was clearly required before proceeding further; this was accomplished by extending the incision obliquely on to the lower abdomen and exposing the main vessels extraperitoneally. It was now seen that the anuerysm did not arise from these vessels, being separated from them by the psoas muscle. Compression of the lower abdominal aorta caused cessation of pulsation within the aneurysm, and it was concluded that it arose from a lumbar

FIG. 2

Excretion pyelograph after aortography showing crushing of the fifth lumbar vertebra, extreme separation of the transverse processes, and displacement of the ureter.

artery. Cautious dissection of the aneurysm was begun, but almost at once heavy haemorrhage occurred, giving a clear demonstration of the mechanism of the recurrent haematoma formation that had characterised his illness. Pressure on the lower aorta enabled the dissection to be completed by peeling away the aneurysm until it was detached from its bed, leaving a posterior shell into which opened a moderate-sized artery that bled copiously when the aorta was released. The feeding artery was identified as the fourth lumbar and was readily controlled by two stitches encircling it. No distal outlet was observed and no further steps were taken to obliterate the aneurysm. A final inspection of the haematoma cavity was made: it was of considerable size, extending from the diaphragm above to the iliac fossa below, with the detached transverse processes projecting as sharp spurs from its lateral wall. A large drain was inserted into the cavity and the incision closed.

The aneurysm formed a rounded mass about two inches across and was composed of firm clot surrounding a comparatively small oval cavity half an inch in its longest diameter. Histological examination confirmed that the wall was thrombus only.

The patient's condition improved dramatically after the operation. He regained good colour and felt much stronger in himself, gaining weight steadily. The wound healed well and recovery of the femoral nerve took place gradually over the next few weeks. A radiograph taken a month after the operation (Fig. 3) showed that the transverse processes had moved back closer to their origins presumably with contraction of the cavity.

FIG. 3
One month after operation.

COMMENT

Traumatic spondylolisthesis is an uncommon injury, but it must be very rare for it to be associated with an aneurysm of a lumbar artery; our search of the literature failed to reveal a similar case. Presumably in our patient a severe closed injury caused laceration of a lumbar artery and this led to the formation of a false aneurysm which leaked intermittently when disturbed by any movement of neighbouring structures. Tears of this or similar arteries must be fairly common with closed injuries but in the great majority spontaneous sealing of the injured artery occurs so that there are no sequelae. Cases such as the one presented here are bound to arise occasionally and it is important that a recurrent massive haematoma should be regarded as a serious condition which may threaten the patient's life if the damaged vessel fails to close spontaneously. Exploration of the haematoma is a major undertaking, and preparations must be made for dealing with any possible arterial injury by resection and grafting should this prove necessary.

ERRORS OF DIAGNOSIS REVEALED AT MENISCECTOMY

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In the years 1940–55 inclusive 2,668 semilunar cartilages were removed by surgeons working as a team and with a similar outlook on the internal derangements of the knee. The detailed records of the cartilages removed show that a relatively small proportion—namely 4·5 per cent—had no detectable lesion. This proportion nevertheless represents 119 cases. The purpose of this paper is to record the results of an investigation of the causes of error, in the belief that the incidence can be reduced.

The term " internal derangement of the knee joint " was first used by William Hey of Leeds in 1784 (Hey 1803), although Hippocrates was familiar with at least one of the conditions concerned. Nothing is known of their surgical treatment until the first record of the removal of a loose body by Paré (1558). It was not until 1877 that a torn semilunar cartilage was removed by Annandale of Edinburgh (Annandale 1885). In contrast, meniscectomy is now perhaps second only to operations for fractures of the neck of the femur on our operation lists.

The same difficulties in diagnosis that troubled the early workers beset us to-day, and normal cartilages continue to be excised in error. Martin (1913) reported an error of 4·5 per cent, Dunn (1934) of 13·66 per cent, Bristow (1935) of 7·2 per cent, and Smillie (1950) of 6·33 per cent. The cases in Smillie's report are included in the present series.

The great majority of cartilages removed show obvious tears, but some have to be closely examined before posterior horn erosions, horizontal cleavage or incomplete splits are revealed. There will however, be no real doubt in the surgeon's mind when he excises a normal cartilage in error. It is also true that experienced surgeons will continue to remove normal structures if damaged ones are not to be missed. This is inevitable in a condition where the reliability of the history is of paramount importance, and where so many other internal derangements exist to confuse.

Figure 1 shows the incidence of error year by year from 1940 to the present day. The increase in errors during the years 1941 to 1946 is explained in great part by the number of unreliable witnesses. With the end of the war many fewer cartilages were removed. At the same time the percentage error was halved. This improvement has been effected by a reduction in the number of malingerers, a careful approach to the problem of localisation, and the increased possibilities for delay in diagnosis. The final organisation of a fully integrated orthopaedic service has in turn brought about a further increase in the total number of cartilage operations performed yearly, but the error has been maintained at the low level of 3 per cent for nine years.

Investigation of the 119 normal cartilages removed shows many causes of error (Table I)

It is clearly of value to examine these errors in detail so that future mistakes may, i. possible, be avoided.

Unreliable witnesses (thirty-two cases)—In this group thirty-two semilunar cartilages were removed in error from twenty-six patients. Of these patients twenty-two were considered to be malingerers (servicemen) or patients interested in compensation for alleged injury. The diagnosis of a torn cartilage depends so much on an accurate history that the surgeon may be misled by a carefully briefed patient, and more so in times of urgency such as a major war. Under normal conditions, however, the surgeon has the opportunity to delay a definite diagnosis, and to refer the patient to a rehabilitation unit to " prove " the knee. Of these twenty-two presumptive malingerers, six had both cartilages excised from the same knee

at one operation. In all these cases doubt regarding the localisation of the lesion existed. Of the remaining sixteen patients six admitted that they had subjected themselves to operation in the knowledge that the joint was normal. Two patients, either before or after the operation in question, parted with all their four cartilages. One patient had had two removed previously and another, whose knee was explored for a retained posterior horn, had a complete intact cartilage removed. Four patients had had one cartilage excised previously, and of these three were known to be normal. All patients of this group gave typical histories of injury, locking, giving way and pain. In all, as might be expected, the anterior cruciate liagment was found to be normal at operation. Nineteen patients gave a history of clicking, but only in six cases was a click elicited by the examining surgeon.

The remaining four patients were all recorded before operation as unreliable witnesses and unable to relax during examination; one patient, in addition, presented a language difficulty.

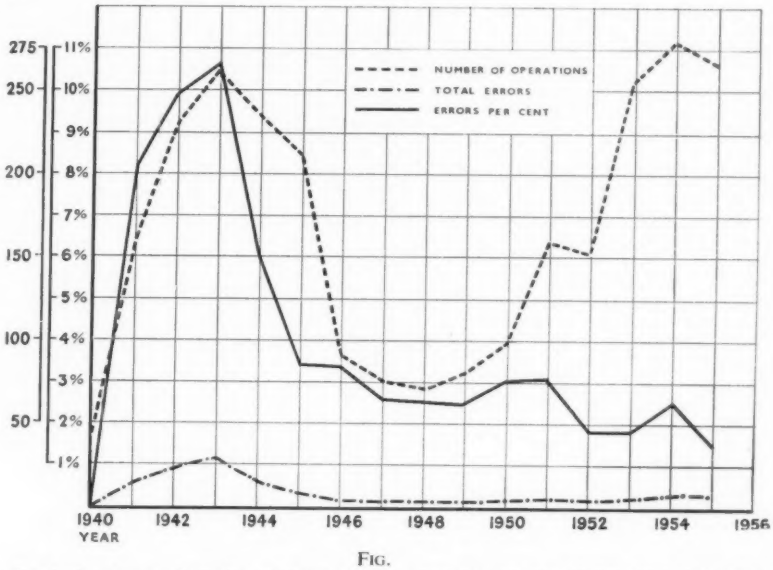

FIG.

Graph showing total number of operations for suspected cartilage lesions from 1940–1955 inclusive, and the incidence of erroneous diagnosis. The percentage error is also shown.

Errors of localisation (twenty cases)—There were twenty known errors of localisation. In two cases both cartilages were thought to be torn, and in a further thirteen difficulty in localisation was encountered before operation. Of the remaining five cases, one had a discoid lateral cartilage and one was found, after excision of the medial cartilage, to have a torn lateral cartilage with a large tag capable of producing locking signs and symptoms on the medial side. Every error of localisation, with one exception, involved the medial cartilage. The sole error with regard to the lateral cartilage was associated with fibrous union of a fractured tibial spine. In half of the cases either a torn (three) or an attenuated (seven) anterior cruciate ligament was found. In five of these cases damage to the cruciate ligament was recognised before operation.

Thirteen of these errors occurred in the six years 1940–45. In the ten years that followed only seven occurred. This reduction was effected by the establishment of a definite approach to the difficult case which is assessed by a close re-examination of the history in the light of the tendency to make errors on the medial side of the joint, and a planned procedure at

operation. The examination of the patient may, of course, often solve the difficulty, particularly if the methods advocated by Sir Robert Jones (1924) and by Fisher (1924) are adopted: extension of the knee is necessary to produce localised pain (Jones) and tenderness (Fisher).

If, despite a clear-cut history, no obvious lesion of the cartilage is revealed through an exploratory incision and the cartilage cannot be drawn forwards with a hook, a separate incision is made on the other side. If again there is no obvious lesion, the first cartilage exposed should be excised. This is necessary because the damage may be confined to the

TABLE I

CAUSES OF WRONG DIAGNOSIS IN 119 CASES OF ARTHROTOMY FOR SUSPECTED CARTILAGE TEARS

Unreliable witness	32
Error of localisation	20
Torn anterior cruciate ligament	9
Quadriceps insufficiency	8
Recurrent subluxation of patella	8
Loose bodies	5
Anterior haemorrhage	4
Chondromalacia patellae	4
Abnormalities of fat pad	3
Strain of medial ligament	2
Slipping semitentinosus tendon	1
Villous arthritis	1
Syphilitic arthritis	1
Sub-sartorial bursa	1
Patello-femoral arthritis	1
Old fracture of tibial condyle	1
Osteochondritis dissecans	1
Unexplained	17

periphery of the posterior horn. Every effort should be made to avoid excising both cartilages unnecessarily, because recovery of function may be slow and incomplete.

The occurrence of damage to the anterior cruciate ligament in half of the errors of localisation calls for note. In all these cases there was a history of an acute injury with much swelling, ill-localised pain and tenderness. Instability appearing later may be explained by the cruciate lesion alone or by later injury to the posterior part of the semilunar cartilage. In this way confusion may arise, especially as momentary locking in the presence of a torn anterior cruciate ligament can happen with a smaller cartilage lesion than is possible otherwise.

Routine air arthrography carried out with meticulous attention to detail, and the interpretation of his own radiographs by a surgeon experienced in the technique, may give a high percentage of accuracy (Somerville 1946). Most surgeons will nevertheless consider that the time involved is not justified. A minimal lesion cannot be demonstrated, and unless in use as a routine procedure arthrography would be valueless in the occasional case of difficulty in localisation. This is the opinion of the author and his colleagues.

Torn anterior cruciate ligament (nine cases)—In nine patients from whom a medial semilunar cartilage was excised in error, isolated lesions of the anterior cruciate ligament were found. In six cases the history was short, varying from one week to one month; the knees were all thought to be locked, with no previous history of instability or locking, and recent tears of the ligament were demonstrated. In two of these examples laxity of the ligament was thought to exist, and examination proved difficult in the other four. This error has occurred only twice since 1945.

In one patient with a history of locking, instability and clicking, the anterior cruciate ligament, in the form of an elongated tag, was hooked forwards from the posterior part of the intercondylar area after excision of a normal cartilage.

In the remaining two patients who presented with instability and medial pain, old-standing damage to the anterior cruciate ligament was found.

Quadriceps insufficiency (eight cases)—Gross wasting of the quadriceps is thought to have been the sole cause of complaints of giving way and pain in eight cases in which a cartilage has been excised in error.

In one patient the obvious effects of old anterior poliomyelitis were to be seen on the other leg, indicating the probability of the cause of the patient's symptoms.

In the other cases incomplete recovery from an injury or operation was thought to be the reason for the quadriceps wasting, and, in turn, of the pain and instability. In three cases previous cartilage operations had been performed three, four and five months before our own exploration. A gunshot wound of the thigh had occurred two months before in two cases, an undisplaced fracture of the tibial condyle eight months before in one case, and an injury two years before, followed by a prolonged stay in hospital, in yet another.

Recurrent subluxation of patella (eight cases)—Seven medial cartilages and one lateral were removed in error when recurrent subluxation of the patella was responsible for the symptoms. On two occasions marked lateral mobility of the patella was noted at operation, and in all of the other cases transplantation of the tibial tubercle was carried out later. Four of these eight errors occurred in females, and these four cases represent half of all the cartilages removed in error from females.

Loose bodies (six cases)—On six occasions no cartilage lesion was demonstrated, but further examination revealed one or more cartilaginous loose bodies. These bodies had not been revealed by previous radiographic examination. In only one patient was the source, in the form of an area of degenerated articular cartilage, discovered at operation. Only one patient gave a history of locking, but all gave a history of injury.

In the absence of positive radiographic findings there was only one example of a frank osteochondritis dissecans lesion as the sole defect.

Anterior haemorrhage (four cases)—In four instances patients were seen with a history of recent injury who, on examination, were considered to have a locked knee but who, at operation, were found to have no lesion of the cartilage but exhibited a large blood clot in the anterior compartment of the joint.

Chondromalacia patellae (four cases)—In two of the four examples of this error there was a clear history of a direct blow, and the records show that tenderness was elicited in the region of the patella. The diagnosis might reasonably have been suspected because there was insufficient evidence to justify a diagnosis of a torn semilunar cartilage.

Abnormalities of the infrapatellar fat pad (three cases)—Fisher (1924) described the anatomy, pathology and symptomatology of anomalies of this structure in some detail, and for some time diagnoses such as "nipping of synovial fringes" and "hypertrophy of the fat pad" have served to disguise a failure to appreciate an underlying cartilage lesion (Smillie 1946). Nevertheless in three cases of the present series the reverse error did occur. On one occasion an enlarged fat pad was thought to be a cystic lateral cartilage, and in another, evidence of a large haemorrhage of some standing was discovered in the fat pad. The third

case presented a typical clinical picture of a discoid lateral cartilage which proved normal on excision. Closer scrutiny revealed that a large part of the infrapatellar fat pad was being trapped between the anterior cruciate ligament and the lateral femoral condyle.

Syphilitic arthritis (one case)—A young soldier, who injured his knee in a fall from a bicycle three weeks before, presented himself with a swollen painful joint, which on examination was thought to be locked. Biopsy unmistakably showed the syphilitic pathology, although macroscopic appearances were not grossly abnormal.

Villous arthritis (one case)—In this example there was a history of injury and a series of recurrent effusions. The joint was considered to be locked, but operation and biopsy demonstrated the true pathology.

Sub-sartorial bursa (one case)—One medial cartilage was removed in the belief that cystic degeneration of the cartilage was present.

Strain of medial ligament (two cases)—In both cases the history was short, and before operation a cartilage lesion was held in considerable doubt.

Patello-femoral arthritis (one case)—The confusion leading to this error arose from the patient's insistence, despite close questioning, that the joint had locked.

Slipping semitendinosus tendon (one case)—The error was caused by an extra-articular exostosis over which the semitendinosus tendon slipped with a loud snap.

Unexplained errors (seventeen cases)—It is thought that at least four cases, all in females, were in fact examples of recurrent subluxation of the patella. No doubt further errors of localisation are still undiscovered, especially if the other side of the joint was not explored in cases in which there was no apparent difficulty in localisation before operation. It may also be that further malingerers have escaped detection.

It must be stated that the initial inexperience of some of the surgeons has played a part in erroneous diagnosis. Malingering is not always easily uncovered, but the experienced will usually recognise discrepancies in the history and histrionics during examination. The knowledgeable surgeon will accept a history of locking of the knee without previous injury in a miner, but refuse to operate if a cartilage click is the only finding in the footballer whose history contains no evidence of instability or locking. Certainly some of the unexplained errors in this series seem to be due to operation in the face of insufficient evidence.

CONCLUSIONS

1. It might be considered that error in 4·5 per cent of meniscectomies is reasonable, but this analysis clearly indicates that many of these mistakes are avoidable. The reduction in error over the past ten years supports this statement.
2. The highest incidence was due to malingering, but this problem is not of great moment under peace-time conditions, which afford opportunity for delay in diagnosis and the use of rehabilitation units.
3. There is an obvious need for careful assessment in cases in which there is doubt about the localisation of the lesion. The marked prevalence of errors on the medial side of the joint, and a planned approach to the operation are of great importance.
4. On fourteen occasions errors were made in cases with a history of recent primary injury, and in each instance the joint was thought to be locked. The emphasis in the differential diagnosis would appear to lean heavily towards lesions of the anterior cruciate ligament and haemorrhage in the anterior part of the joint. This serves to emphasise that great care is required in the assessment of recent injuries. A more cautious attitude is necessary, and, when the integrity of the anterior cruciate ligament is in doubt, examination under anaesthesia is always justifiable and sometimes necessary.
5. Recurrent subluxation of the patella is a source of error that should be noted, especially in women. Smillie (1950) stated that " errors of diagnosis occur most frequently in the female. It is hardly an exaggeration of the situation to suggest that every internal derangement

of the knee joint in a woman should be suspected to be a recurrent subluxation of the patella until proved otherwise; tears of the meniscus are rare."

6. Caution must be the keynote when gross quadriceps wasting is the main feature. The surgeon must ensure that as good a recovery as possible has been made after previous injury before further operation is contemplated.

SUMMARY

1. In the course of 2,668 operations for suspected cartilage lesions 119 errors of diagnosis were revealed.

2. These errors are analysed and certain conclusions are drawn.

My thanks are due to my senior colleague, Mr I. S. Smillie, Director of Orthopaedics for the Eastern Region of Scotland. Much of the material used in this investigation has already been the subject of his widespread researches into the traumatic pathology of the knee joint. Mr Smillie has always been and remains a most stimulating mentor, and I cannot overstress my gratitude to him. I would like also to thank other colleagues, past and present, for the use of their clinical material.

REFERENCES

ANNANDALE, T. (1885): An Operation for Displaced Semilunar Cartilage. British Medical Journal, **i**, 779.

BRISTOW, W. R. (1935): Internal Derangement of the Knee Joint. Journal of Bone and Joint Surgery, **17**, 605.

DUNN, N. (1934): Observations on Some Injuries of the Knee-joint. Lancet. **i**, 1,267.

FISHER, A. G. T. (1924): Internal Derangements of the Knee-joint, pp. 16, 41 and 127. London: H. K. Lewis & Co. Ltd.

HEY, W. (1803): Practical Observations in Surgery, p. 327. London: T. Cadell and W. Davies.

HIPPOCRATES (1849): The Genuine Works. Translated by F. Adams. Volume 2. London: The Sydenham Society.

JONES, Sir R. (1924): Note 5 in A. G. T. Fisher's Internal Derangements of the Knee-joint, p. 189.

MARTIN, A. M. (1913): Injuries to Semilunar Cartilages. Proceedings of the Royal Society of Medicine, Surgical Section, **6**, 23.

PARÉ, A. (1841): Oeuvres Complètes. Tome 3, p. 32. Paris: J. B. Baillière.

SMILLIE, I. S. (1946): Injuries of the Knee Joint, p. 287. Edinburgh: E. & S. Livingstone Ltd.

SMILLIE, I. S. (1950): Injuries of the Knee Joint. Second edition, pp. 101, 250. Edinburgh: E. & S. Livingstone Ltd.

SOMERVILLE, E. W. (1946): Air Arthrography as an Aid to Diagnosis of Lesions of the Menisci of the Knee Joint. Journal of Bone and Joint Surgery, **28**, 451.

ANTERIOR DISLOCATION OF THE HEAD OF THE RADIUS ASSOCIATED WITH UNDISPLACED FRACTURE OF THE OLECRANON IN CHILDREN

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Anterior dislocation of the head of the radius is rare. Congenital and traumatic varieties are described. McFarland (1936) has described certain characteristics which distinguish the congenital variety. Traumatic dislocation occurs most commonly in the extension type of Monteggia fracture-dislocation. It has been described as part of complex fracture-dislocations of the elbow joint, and as an isolated injury. Dislocation occurs also in diaphysial aclasis and dyschondroplasia in association with defective growth of the ulna (Fairbank 1951).

Three cases are described of anterior dislocation of the head of the radius with undisplaced fracture of the olecranon.

CASE REPORTS

Case 1—A boy aged six years fell off his scooter injuring his left elbow. Radiographs showed an oblique fracture of the olecranon with forward dislocation of the radial head (Fig. 1). The dislocation was reduced under general anaesthesia, and plaster immobilisation was maintained for six weeks. Reduction was maintained (Fig. 2). Two weeks later he had regained a full and painless range of both elbow and radio-ulnar movements, and radiographs were indistinguishable from normal.

Case 2—A girl aged seven years fell with her right elbow under her and sustained a transverse fracture of the olecranon with forward dislocation of the radial head (Fig. 3). This was reduced under general anaesthesia. The elbow was re-examined two days later under anaesthesia and found to be stable with the elbow flexed to a right angle and the forearm supinated. A plaster was retained for six weeks. One month later the elbow was indistinguishable from the normal. A radiograph six months after injury is shown in Figure 4.

Case 3—A girl aged seven years fell with her left arm under her and sustained a transverse fracture of the olecranon with forward dislocation of the radial head (Fig. 3). The dislocation was not observed initially and the injury was treated as an uncomplicated fracture of the olecranon with the elbow in extension. Two weeks later a review of the radiographs revealed the dislocation of the radial head, and further films showed a mass of new bone lying in front of the displaced head. The dislocation was reduced with some difficulty under general anaesthesia (Fig. 4), and plaster immobilisation was maintained for four weeks. Since then there has been a gradual diminution in the myositis ossificans and a steady increase in the range of elbow and radio-ulnar movements.

DISCUSSION

Occurrence—A review of the literature has failed to reveal any description of this injury. It was mentioned, but not described, by Smith (1954) and by Blount (1954). Speed (1935) described a hyperextension injury to the elbow with displacement of the olecranon epiphysis and forward dislocation of the radial head. In a series of 439 elbow injuries Wilson (1933) found only three cases with anterior dislocation of the head of the radius and all were associated with displaced fractures of the shaft of the ulna.

Characteristics—The olecranon fracture was clinically inconspicuous; the line of the fracture was transverse in two cases and oblique in one.

Swelling and tenderness over the displaced radial head were the most conspicuous clinical feature, but, although they were present, the dislocation was not noticed in Case 3.

FIG. 1
Case 1—Oblique fracture of the olecranon with anterior dislocation of the radial head.

FIG. 2
Case 1—After reduction.

FIG. 3
Case 2—Transverse fracture of the olecranon with anterior dislocation of the radial head.

FIG. 4
Case 2—After reduction.

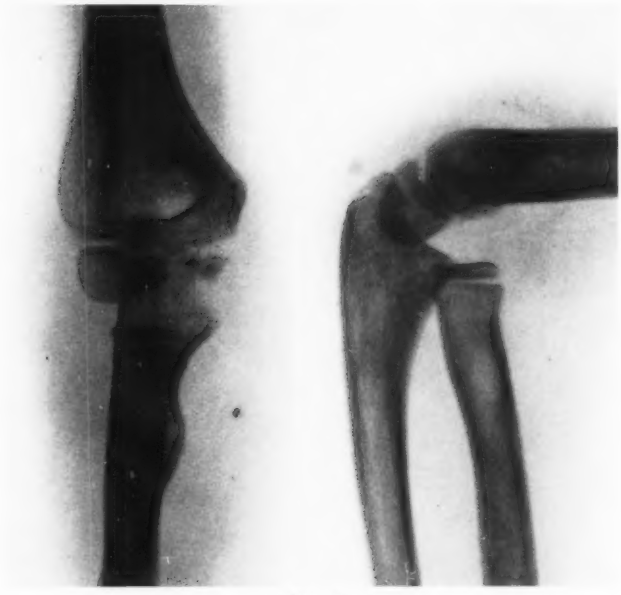

FIG. 5
Case 3—Injury like that in Case 2.

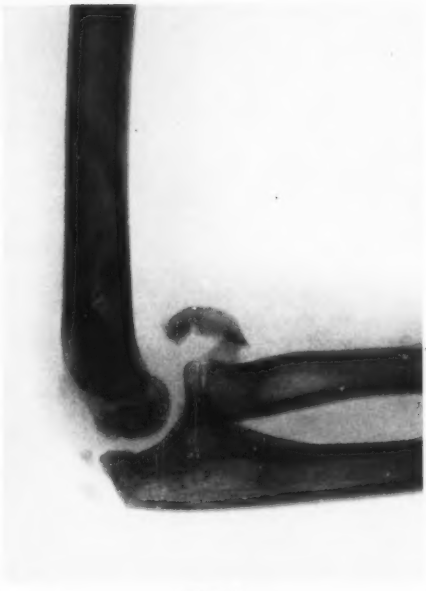

FIG. 6
Case 3—Radiograph after late reduction of the dislocated radial head, showing myositis ossificans in the former position of the displaced head.

The rapid development of myositis ossificans in front of the displaced radial head that occurred in this case serves to emphasise the importance of an early and accurate diagnosis.

Treatment—The dislocations were reduced under general anaesthesia by supination of the forearm and were stable in the position of supination with the elbow flexed to a right angle. Immobilisation in plaster was maintained for six weeks, the child then being allowed to regain elbow movements unaided.

Mechanism of the injury—No clear picture of this could be obtained, but it appeared that in each case the child fell heavily with the arm under the body. Corbett (1931) ascribed a case of anterior dislocation of the head of the radius to a combination of pronation of the forearm and hyperextension of the elbow, and Evans (1949) has emphasised the effects of pronation in a fall on the outstretched hand as a mechanism of the Monteggia fracture-dislocation. It is suggested that in the present cases the fracture of the olecranon is the result of a hyperextension injury to the elbow and that the forward dislocation of the radial head is produced by a concomitant pronation of the forearm.

SUMMARY

1. Three cases of anterior dislocation of the head of the radius associated with an undisplaced fracture of the olecranon are described.

2. A combination of pronation of the forearm and hyperextension of the elbow is suggested as a possible mechanism.

I wish to thank Mr O. J. Vaughan-Jackson and Mr P. G. Epps for permission to publish these cases and for their help and advice in the preparation of this paper.

REFERENCES

BLOUNT, W. P. (1954): Fractures in Children. Baltimore: The Williams & Wilkins Company.

CORBETT, C. H. (1931): Anterior Dislocation of the Radius and its Recurrence. British Journal of Surgery, **19,** 155.

EVANS, E. M. (1949): Pronation Injuries of the Forearm. Journal of Bone and Joint Surgery, **31-B,** 578.

FAIRBANK, Sir T. (1951): An Atlas of General Affections of the Skeleton. Edinburgh and London: E. & S. Livingstone Ltd.

MCFARLAND, B. (1936): Congenital Dislocation of the Head of the Radius. British Journal of Surgery, **24,** 41.

SMITH, F. M. (1954): Surgery of the Elbow. Springfield, Illinois: Charles C. Thomas.

SPEED, K. (1935): A Text-book of Fractures and Dislocations. Third edition. London: Henry Kimpton.

WILSON, P. D. (1933): Fractures and Dislocations in the Region of the Elbow. Surgery, Gynecology and Obstetrics, **56,** 335.

AINHUM

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As the number of coloured immigrants into the United Kingdom increases we must expect to see more examples of the few diseases that are peculiar to negroes. Among these is ainhum. In some tropical areas this is by no means a rare disease, for there were over 200 cases recorded in medical literature before the second world war. So far, however, only one case has been reported in the British Isles. We are reporting a second, study of which has enabled us to throw a little new light upon the pathology of the condition.

This interesting disease is confined to the toes, to negroes and, almost exclusively, to the male sex. There are no reports of indisputable cases in females except that of Goehring (1944), and none at all in half-castes, in Indians, or in other dark-skinned peoples. The large majority of true cases of ainhum have occurred in patients aged between twenty and fifty years.

There is some doubt about the origin of the name. It is usually thought to be derived from the Nago or Yoruba word for " to saw " or " to cut " (Castellani and Chambers 1919).

The clinical picture is unmistakable. A constricting sulcus appears around the base of one fifth toe and later, usually, around that of the other. The sulcus deepens, the toe becomes progressively externally rotated, its connection to the foot becomes increasingly slender, and if it is not amputated it eventually drops off.

CASE REPORT

A West African negro, born in Lagos in or about 1911, was transferred to the Manchester Royal Infirmary from the Manchester and Salford Hospital for Skin Diseases in March 1956. He had lived in Lagos until 1944, when he became a merchant seaman and visited Britain for the first time. At no time had he gone barefoot and he had always lived in cities. In 1947 he settled in Manchester as a café proprietor, and he has not left England since.

In 1954 he noticed a shallow, linear groove in the skin of the dorsal surface of the base of his fifth right toe and, shortly afterwards, of his left fifth toe also. On the right side the groove became progressively deeper, on the left less so. After about two years the right toe gave him some pain.

He had had no important previous illnesses. He gave no family history of similar trouble. He had, in fact, never heard of ainhum.

The state of his feet on admission to hospital is shown in Figure 1. The sulcus around the right fifth toe appeared mildly inflamed. It encircled the toe at the level of the proximal end of the middle phalanx. Distal to the sulcus the toe was swollen and the skin of its plantar surface had a glazed appearance. The groove on the left toe was shallower and did not quite encircle the toe on the plantar surface. Both fifth toes were warm. Sensation in them was normal and, though voluntary movement was impossible on the right side, it was performed fully on the left.

There was a scaly patch on each side of his neck. These patches had been unchanged for twenty years, and were neither infiltrated nor anaesthetic. No other lesions could be found.

The blood was normal. The Wassermann reaction was negative. Scrapings from the sulci and from between the other toes did not produce any fungus, neither did scrapings from the lesion of the skin of the neck. Culture of a smear taken from the inflamed sulcus on the right fifth toe grew a staphylococcus, sensitive to chloramphenicol and to erythromycin.

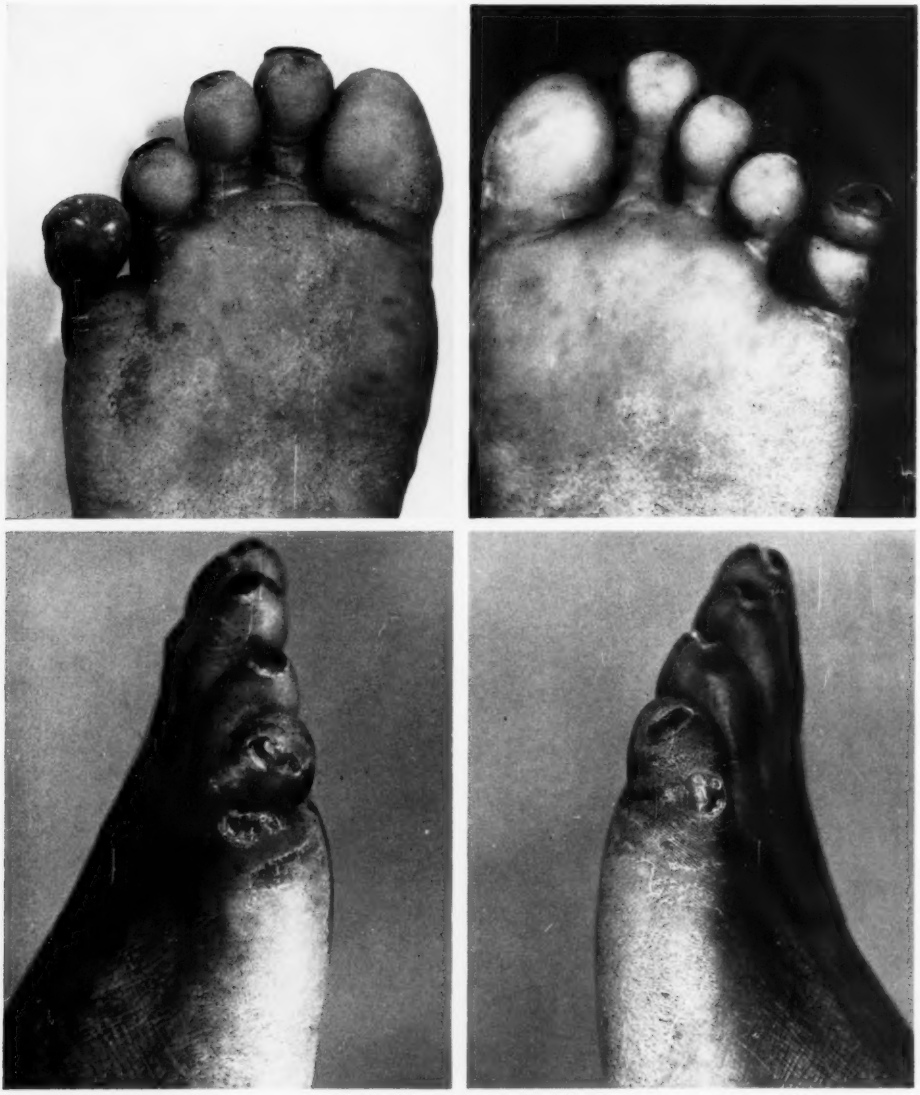

FIG. 1

The right and left feet of the patient described, a forty-five-year-old negro suffering from ainhum. Note the grooves around the fifth toes, the lateral rotation of the affected toes and the asymmetrical involvement of them.

Radiographs (Fig. 2) showed that the sulcus on the right side was almost amputating the fifth toe in the region of the proximal interphalangeal joint. The appearances were those of erosion of the head of the proximal phalanx. The bony outlines were normal on the left

side. On both sides, the middle phalanges, which are the bones most commonly attacked by ainhum (Spinzig 1939), looked normal. These appearances are shown more clearly in the radiograph of the amputated toe (Fig. 3).

FIG. 2
Radiographs of the feet.

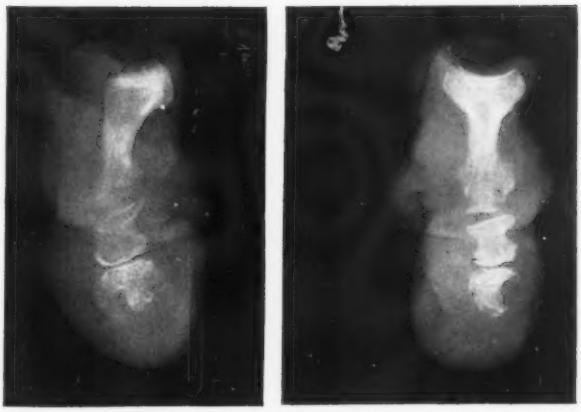

FIG. 3
Radiographs of the amputated right fifth toe.

The right fifth toe was amputated through the metatarso-phalangeal joint. The cut surfaces bled normally and healed well. The patient refused any form of treatment for the left fifth toe.

PATHOLOGY

Histological sections of the whole toe were taken in a plane running longitudinally through the nail, and in a plane at right angles to this surface. The constriction lay at the level of the proximal end of the middle phalanx and was deeper on the volar aspect, where the sulcus contained much keratinous debris (Fig. 4). The epidermis covering it was intact at the three sites examined, though it was compressed at the apex of the sulcus. In the region of the constriction, the zone between the epidermis and the bone or the joint capsule consisted of two distinct layers. The more superficial of these layers was relatively free from inflammatory infiltration; the morphology and orientation of its fibre bundles were like those of normal dermal tissue. The deep subcutaneous layer, by contrast, was oedematous, deficient in fat and infiltrated with plasma cells and with lymphocytes. These cells were particularly related to areas of perivascular oedema. The fibre bundles in the deeper tissues, though disrupted here and there by cellular infiltration, were mainly orientated longitudinally (Fig. 5). Numerous gram-negative diplococci or cocco-bacilli were found in a small area of the granulation tissue deep in the volar subcutaneous tissues just proximal to the sulcus. The significance of this finding is obscure because organisms were not found in other parts apparently equally severely affected.

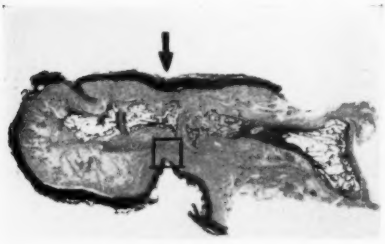

FIG. 4

Longitudinal section of the right fifth toe. The arrow marks the site of the constriction. The area outlined is reproduced in Figure 5.

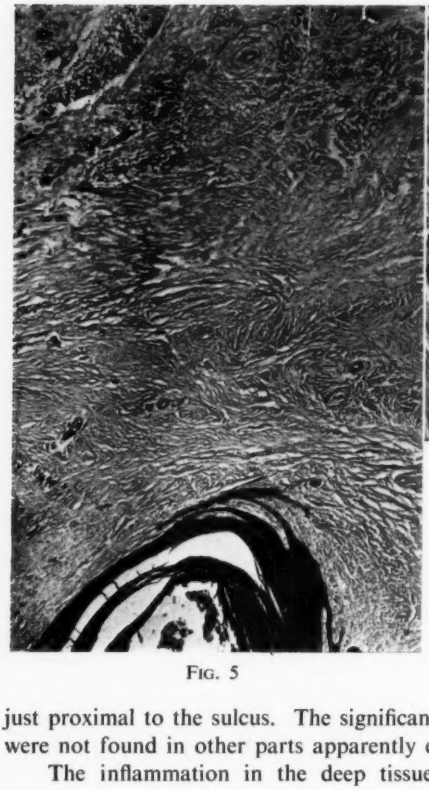

FIG. 5

FIG. 6

Figure 5—Higher power view of the area indicated in Figure 4. Note, from below upwards, the apex of the sulcus, normal dermis, and chronically inflamed deep tissues. Figure 6—High power view of the proximal interphalangeal joint, showing inflammatory infiltration of the capsule anderosion of the articular cartilage.

The inflammation in the deep tissues was greatest in the region of the proximal

interphalangeal joint, but it extended from the metatarso-phalangeal joint distally as far as the terminal joint.

Both interphalangeal joints and, to a lesser extent, the metatarso-phalangeal joint, were involved in a chronic inflammatory process apparently continuous with that in the deep tissues. This resulted in an erosive arthropathy histologically indistinguishable from that seen in rheumatoid arthritis (Fig. 6). Inflammatory change also involved the periosteum; new periosteal bone had formed on both phalanges (Fig. 4).

DISCUSSION

Ainhum was first described in 1860 by Clarke, " late Surgeon to the Natives " of the Gold Coast. He called it " dry gangrene of the little toe of negroes." It was first recognised as a distinct disease by da Silva Lima of Bahia in 1867, and his account of the clinical features has not yet been surpassed. The disease has been reported in all parts of West Africa, in the West Indies (Doyle 1889, Jiménez 1946), in Panama (Kean and Tucker 1946, Kean, Tucker and Miller 1946), in South America and at least fifty times in the United States of America, mostly in the Southern States (Spinzig 1939, McKnight 1940, Montgomery 1942, Spencer 1942, Cornbleet 1946, Hersh 1946, Omens 1946, Tye 1946, Goehring 1944, Hucherson 1950 and Stack 1950). Several of the reported cases have occurred in negroes born in the United States. Indeed Matas (1896) stated that it was " not prevalent " among the African slaves originally imported into that country.

The disease was seen in a Krooman serving in the Royal Navy in 1917 (Barton 1918) and in a fifty-five-year-old negro in Bristol in 1941 (Davies and Hewer 1941). We have not found any other report of a case in the United Kingdom.

The fifth toe has been affected in all reported cases, and in most instances both fifth toes. It is unusual for the disease to reach the same stage in each fifth toe at the same time. Involvement of the fourth toes may rarely follow the development of the disease in the fifth; in a very few cases (Kean, Tucker and Miller 1946) there has been involvement of the third, fourth and fifth toes of both feet.

A strong family history has been given by some patients (Duhring 1884, Simon 1921, Weinstein 1912).

Ainhum has been much confused with other diseases. Such confusion accounts for the reports of its occurrence in women and girls, in white persons, or in the hands (Wigley 1929, Drummond 1939, Johnson 1941, Neumann 1953, Priesel 1949, Sutton and Sutton 1935, Findlay 1951, Wells and Robinson 1952, Bluefarb 1948). There is no doubt that these reports in fact describe cases of congenital annular constriction of digits. Confusion with leprosy (Montestruc and Caubet 1947) is perhaps to be expected, for ainhum has been reported in persons suffering also from leprosy (Matas 1896). However, none of the forty-five cases considered by Kean *et al.* showed any evidence of leprosy, and none of the 110 negro lepers whom they studied post-mortem had ainhum. At least nine of the reported cases of ainhum had positive Wassermann reactions (Wright 1924, Kean *et al.* 1946), but the large majority of the others showed no evidence of syphilis. One case of alleged ainhum (Shaffer 1947) was almost certainly an example of diabetic gangrene.

The disease has occurred in sufferers from peripheral vascular disease (Davies and Hewer 1941) and in victims of syringomyelia (Tatz 1946). It has been regarded on rather scanty evidence as a variety of scleroderma (Acton 1928). It is often accompanied by hyperkeratosis of the plantar skin (Wigley 1929, Drummond 1939, Spencer 1942).

Ainhum is certainly not due to self-mutilation, nor to any form of injury. It occurs in negroes who have never gone bare-foot and in others who have never worn boots or shoes. It does not occur in non-negro people who dispense with footwear, even if they live in jungles. It is not due to infestation by parasites, as suggested by Wellman (1906).

THE NATURE OF THE DISEASE

Accounts of the histology of ainhum have been contradictory. Wright (1924) found evidence of endarteritis, but the Wassermann reaction was positive in his patient. Davies and Hewer (1941), who gave a full description of the histology in their case, also found thickening of the walls of the arteries and narrowing of their calibre, but their patient had generalised peripheral vascular disease. Ash and Spitz (1945) did not find any significant change in the vessels or in the nerves in a patient free from generalised arterial disease. Martens and Norris (1945) found evidence of osteomyelitis, and suggested that ainhum begins as such. Kean and Tucker (1946), on the other hand, found neither arterial disease nor osteomyelitis.

In our case, histological evidence indicates that the primary lesion is a chronic, non-specific and widespread concentric inflammation of the deep fascial layers and of the peritendinous structures of the toes. The erosive arthropathy and the periostitis can be explained as extensions of this process. The constricting band is apparently due to a purse-string effect of the deep inflammation on the normal dermal tissues. We cannot suggest how this effect is produced, or why the constriction usually appears in much the same site on every affected toe, though the site of the sulcus, which is not entirely free from variation, may merely indicate the site of origin of the inflammatory reaction. It certainly seems more likely that the clinical features depend on the curious anatomy of the inflammation rather than upon any unique etiological agent or unusual tissue response.

REFERENCES

Acton, H. W. (1928): Ainhum, a Band Scleroderma. Indian Journal of Medical Research, **15,** 1,085.

Ash, J. E., and Spitz, S. (1945): Pathology of Tropical Diseases, p. 344. Philadelphia and London: W. B. Saunders Company.

Barton, S. S. (1918): A Case of Ainhum. Journal of the Royal Naval Medical Service, **4,** 422.

Bluefarb, S. M. (1948): Constriction of the Finger Simulating Ainhum. Archives of Dermatology and Syphilology, **57,** 741.

Castellani, A., and Chambers, A. J. (1919): Manual of Tropical Medicine. Third edition. London: Baillière, Tindall and Cox.

Clarke, R. (1863): Remarks on the Topography and Diseases of the Gold Coast. Transactions of the Epidemiological Society of London, **1,** 76.

Cornbleet, T. (1946): Ainhum. Archives of Dermatology and Syphilology, **54,** 628.

Davies, J. N. P., and Hewer, T. F. (1941): Ainhum: Report of a Case in England with Histological Study. Transactions of the Royal Society of Tropical Medicine and Hygiene, **35,** 125.

Doyle, E. A. G. (1889): Ainhum. British Medical Journal, **i,** 1,346.

Drummond, M. (1939): A Case of Unusual Skin Disease. Irish Journal of Medical Science, 85.

Duhring, L. A. (1884): A Case of Ainhum. American Journal of the Medical Sciences, **87,** 150.

Findlay, G. H. (1951): Ainhum: with special reference to Involvement of the Fingers. Transactions of the Royal Society of Tropical Medicine and Hygiene, **44,** 747.

Goehring, W. O. (1944): Ainhum. Pennsylvania Medical Journal, **47,** 1,089.

Hersh, J. (1946): Ainhum: Report of a Case. Journal of the International College of Surgeons, **9,** 477.

Hucherson, D. C. (1950): Ainhum (Dactylolysis Spontanea). A Review of Ten Cases. Annals of Surgery, **132,** 312.

Jiménez, J. de J. (1946): El ainhum en la República Dominicana. Boletin de la Asociación Médica de Santiago, **4,** 55.

Johnson, H. M. (1941): Congenital Cicatrizing Bands. American Journal of Surgery, **N.S. 52,** 498.

Kean, B. H., and Tucker, H. A. (1946): Etiologic Concepts and Pathologic Aspects of Ainhum. Archives of Pathology, **41,** 639.

Kean, B. H., Tucker, H. A., and Miller, W. C. (1946): Ainhum: A Clinical Summary of Forty-five Cases on the Isthmus of Panama. Transactions of the Royal Society of Tropical Medicine and Hygiene, **39,** 331.

McKnight, R. B. (1940): A Case of Ainhum Treated by Lumbar Sympathetic Ganglionectomy. North Carolina Medical Journal, **1,** 76.

Martens, V. E., and Norris, R. F. (1945): Pathologic Findings in a Case of Ainhum. United States Naval Medical Bulletin, **45,** 745.

Matas, R. (1896): The Surgical Peculiarities of the Negro. Transactions of the American Surgical Association, **14,** 483.

MONTESTRUC, E., and CAUBET, P. (1947): Ainhum in a Leper. Transactions of the Royal Society of Tropical Medicine and Hygiene, **41,** 187.
MONTGOMERY, R. M. (1942): Ainhum. Archives of Dermatology and Syphilology, **45,** 400.
NEUMANN, A. (1953): Pseudoainhum. Archives of Dermatology and Syphilology, **68,** 421.
OMENS, D. V. (1946): Ainhum. Archives of Dermatology and Syphilology, **54,** 238.
PRIESEL, R. (1949): Daktylolysis Spontanea Ainhumoides. Österreichische Zeitschrift für Kinderheilkunde und Kinderfürsorge, **3,** 107.
SHAFFER, L. J. O. (1947): Ainhum. Archives of Pathology, **43,** 170.
DA SILVA LIMA, J. F. (1867): Estudo sobre o " Ainhum." Gazeta Medica da Bahia, **1,** 146, 172.
SIMON, K. M. B. (1921): Ainhum, a Family Disease. Journal of the American Medical Association, **76,** 590.
SPENCER, G. A. (1942): Ainhum Associated with Hyperkeratosis Palmaris et Plantaris. Archives of Dermatology and Syphiliology, **45,** 574.
SPINZIG, E. W. (1939): Ainhum: Its Occurrence in the United States. With a Report of Three Cases. American Journal of Roentgenology, **42,** 246.
STACK, J. K. (1950): Ainhum. Journal of Bone and Joint Surgery, **32-A,** 444.
SUTTON, R. L., and SUTTON, R. L., Jun. (1935): Diseases of the Skin. Ninth edition, p. 624. London: Henry Kimpton.
TATZ, K. (1946): Pityriasis Rubra Pilaris with Ainhum and Syringomyelia. British Journal of Dermatology and Syphilis, **58,** 123.
TYE, M. (1946): Ainhum. New England Journal of Medicine, **234,** 152.
WEINSTEIN, H. (1912): A Description of Ainhum with Report of Interesting Cases Occurring in One Family. Proceedings of the Canal Zone Medical Association, **4,** 110.
WELLMAN, F. C. (1906): A Criticism of Some of the Theories Regarding the Etiology of Goundou and Ainhum. Journal of the American Medical Association, **46,** 636.
WELLS, T. L., and ROBINSON, R. C. V. (1952): Annular Constrictions of the Digits. Archives of Dermatology and Syphilology, **66,** 569.
WIGLEY, J. E. M. (1929): A Case of Hyperkeratosis Palmaris et Plantaris associated with Ainhum-like Constriction of the Fingers. British Journal of Dermatology and Syphilis, **41,** 188.
WRIGHT, L. T. (1924): Ainhum. Report of a Case Occurring in a Man with Syphilis. Urologic and Cutaneous Review, **28,** 135.

PARTIAL CUBO-NAVICULAR COALITION AS A CAUSE OF PERONEAL SPASTIC FLAT FOOT

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It is now recognised that congenital fusion between the talus and the calcaneum or between the calcaneum and the navicular bone is found in a high proportion of patients who have peroneal spastic flat foot. This paper presents an example of partial coalition between the cuboid and navicular bones.

In the normal foot there may or may not be a synovial joint between these two bones. Oblique radiographs show well defined articular facets in approximately one quarter of the feet examined. In the remainder there is a space between the adjacent angles of the cuboid and navicular, the edges of the bones being irregular. It is important to appreciate that slight alterations in the obliquity of the x-rays can produce an overlapping of shadows which may suggest bony fusion. Further films, at varying angles, will demonstrate the true situation.

Harris and Beath (1948) suggested that the typical congenital tarsal fusions occur in relation to accessory bones. The os cuboidum secundarium is found on the plantar surface of the foot at the point of contact between cuboid, navicular, calcaneum and talus (Trolle 1948). It may be fused to the navicular or cuboid, but more commonly to the former (O'Rahilly 1953). Jack (1954) suggested that coalition was due to an error in differentiation (possibly hereditary) which may lead to a large complete bony fusion at one extreme or to a small accessory bone at the other. Since an accessory bone may be present between the navicular and the cuboid, it is reasonable to suppose that coalition may occur between the two bones. Dwight (1907) illustrated by radiograph and photograph of the plantar surface of the bones of the foot what he described as synchondrosis of the navicular and cuboid, but it has not been possible to find any account of this particular abnormality as a cause of spastic flat foot.

CASE REPORT

The patient, a boy aged fifteen years when first seen, complained of pain in his left foot. There was no history of injury. The pain had occurred during the previous two weeks with standing and walking. On examination, both feet were valgus, with considerable peroneal spasm in the left foot which prevented supination. This movement was present on the right side and there was no spasm. Radiographs showed that there appeared to be a bridge of bone between the navicular and the cuboid in the left foot (Fig. 1) and a partial coalition between these bones in the right foot (Fig. 2). Later, tomographs were done which showed that the bridge on the left side was also incomplete (Fig. 3).

At first it had been impossible to correct the valgus deformity of the left foot, but after a few days' rest it was found that the peroneal spasm could be relaxed by gentle pressure and full supination was possible. Once this had happened the foot was completely mobile. If the boy was then made to walk a short distance the spasm returned, but could again be overcome. The foot was immobilised in the corrected position in a below-knee walking plaster for six weeks. He was then given an outside iron and inside T-strap, and later a rigid arch support was made to a cast of the foot. These measures failed to control the peroneal spasm (which can still be relaxed after a period of rest), but since he has very little pain more active treatment has not been advised. He has been able to begin work as a cabinet maker.

Radiographs were also taken of the feet of the boy's father, mother and sister, but these did not show any abnormality.

FIG. 1

Antero-posterior and oblique radiographs of left foot showing what appears to be a complete bridge between the cuboid and navicular.

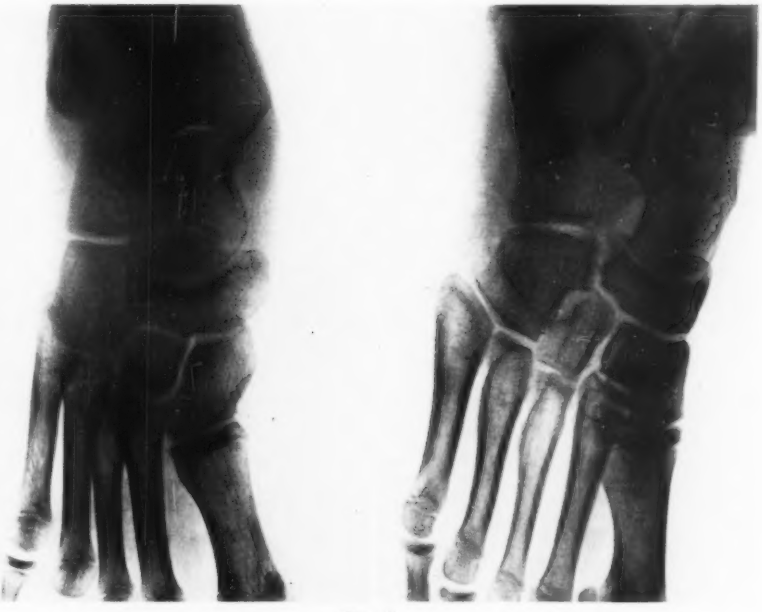

FIG. 2

Antero-posterior and oblique radiographs of right foot showing an incomplete cubo-navicular coalition.

DISCUSSION

The cause of pain in patients with calcaneo-navicular and talo-calcaneal bars has been the subject of controversy. When the bar forms a solid bridge, it is suggested that the interference with the normal smooth action of the subtalar-midtarsal joints explains the clinical picture (Seddon 1932). The additional strain thus thrown on the normal joints may result in osteoarthritic changes. This is typically represented by lipping of the upper part of the talo-navicular joint which Harris and Beath (1948) regarded as almost indicative of a congenital tarsal fusion.

FIG. 3
Oblique tomograph of left foot (4·5 centimetres cut) showing the bridge between cuboid and navicular and demonstrating that it is not complete.

In this case it seems unlikely that partial coalition between the navicular and the cuboid could sufficiently derange the mechanics of the foot to produce pain in the normal peritalar joints. It is, therefore, reasonable to suppose that the pain might arise directly from the site of the abnormal fusion. It was localised to this region and was not spread widely over the foot. When the coalition is incomplete, movement in the involved joint is limited; consequently the union is particularly liable to strains and so may give rise to symptoms. Furthermore, Harris (1956) stated that in his experience incomplete coalition in the calcaneo-navicular bar and talo-calcaneal bridge was more apt to cause pain that a solid bony fusion.

The absence of pain in the opposite foot is not easy to explain, although the same may happen in patients with bilateral incomplete calcaneo-navicular bars. It can only be presumed that the symptoms are set off by an initial acute injury, or that the fibrous union is more lax in this foot (as is suggested by the slightly wider gap seen in the radiographs) and so less liable to strain.

There is no doubt that in this case there was true peroneal spasm rather than adaptive shortening, because the position of the foot could be fully corrected by gentle pressure. The spasm is probably a non-specific protective reaction to pain, but there is no clear reason why the pronators should be involved. The mechanism described by Lapidus (1946), which implied a lesion of the interosseous talo-calcaneal ligament or subtalar joint, is not applicable.

Although the cubo-navicular coalition must be a very uncommon abnormality it should be looked for carefully in cases of spastic flat foot which do not show an obvious structural anomaly. The cubo-navicular angle should be studied in oblique radiographs, and if the normal gap or joint cannot be successfully demonstrated in a single view further plain films or tomographs will be necessary.

My thanks are due to Professor J. Trueta for his help in preparing this report for publication, and to Dr R. I. Harris for his advice and encouragement.

REFERENCES

DWIGHT, T. (1907): Variations of the Bones of the Hands and Feet. Philadelphia and London: J. B. Lippincott Company.

HARRIS, R. I., and BEATH, T. (1948): Aetiology of Peroneal Spastic Flat Foot. Journal of Bone and Joint Surgery, **30–B**, 624.

HARRIS, R. I. (1956): Personal communication.

JACK, E. A. (1954): Bone Anomalies of the Tarsus in Relation to "Peroneal Spastic Flat Foot." Journal of Bone and Joint Surgery, **36–B**, 530.

LAPIDUS, P. W. (1946): Spastic Flat-foot. Journal of Bone and Joint Surgery, **28**, 126.

O'RAHILLY, R. (1953): A Survey of Carpal and Tarsal Anomalies. Journal of Bone and Joint Surgery, **35–A**, 626.

SEDDON, H. J. (1932): Calcaneo-scaphoid Coalition. Proceedings of the Royal Society of Medicine (Section of Orthopaedics), **26**, 419.

TROLLE, D. (1948): Accessory Bones of the Human Foot, p. 26. Copenhagen: Munksgaard.

MULTIFOCAL OSTEOGENIC SARCOMA

Report of a Case

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Multiple osteogenic sarcomata are rare tumours of the skeleton, apart from those cases associated with Paget's osteitis deformans. The case presented here resembles in certain ways those already reported, but it differs in the age of the patient, the relatively slow growth of the skeletal tumours, and in the absence of pulmonary metastases. Moreover, the individual tumour masses can be recognised as being akin to unifocal osteoblastic osteogenic sarcoma of low grade (Busso and Schajowicz 1945–46). The extensive bone involvement seen in this patient was noted radiologically at the time of her first examination, five or six months after the date of the presenting symptom.

CASE REPORT

The patient was a married woman aged fifty, with one daughter. Her periods had been irregular since the age of forty, but of normal duration and volume; they ceased at the age of fifty. When she was fifty the patient was treated with ethinyl oestradiol (1 milligram three times a day), and later with testosterone to a total amount of 100 milligrams. The patient had always had a dark skin, which was said to be familial; but pigmentation around the vulva had recently increased. When she was forty-two a "naevus" was removed from the skin of the left groin.

When first seen in December 1950 the patient complained of progressive pain in her right knee of five to six months' duration. This did not respond to varied treatment and was soon followed by low back pain. At the same time a sternal swelling (of unrecorded duration), previously stationary in size, began to grow. More recently she had noted a further swelling of the right parietal bone. A fall in August 1950 was followed by swelling of the left knee, with intractable aching and weakness of the legs. There had been no loss of weight.

On examination the general condition was fairly good. The blood pressure was 170/90. There were no palpable lymph nodes. A gynaecological examination revealed no abnormality. *Skin*—The whole body, face and extremities showed generalised dark brown pigmentation, especially marked at the nipples, vulva and extensor surfaces of the forearms.

Musculo-skeletal system—There was no pain when at rest, but movement was painful and limited, especially of the left knee and spine. Muscle tone and reflexes were normal in the upper limbs. In the lower limbs muscle tone was increased, but power was normal. The left patellar and ankle jerks and the left plantar response were absent. There was no sensory change. There was a smooth hard mass (10 × 10 centimetres) attached to the upper end of the sternum; this was not tender on pressure. There were two similar tumours arising from the right parietal bone and the occiput.

Radiological examination—The bones involved were the skull, sternum, right humerus and scapula, right first rib and most of the vertebrae, and the pelvis and femora. *Skull:* There was a dense lesion in the occipital bone 9·5 centimetres long by about 10 centimetres wide and 5 centimetres thick. In the right posterior parietal area there was another oval mass 6·5 centimetres by 3·5 centimetres and 1 centimetre thick. The rest of the calvarium appeared to be of normal texture and was about 0·8 centimetre in thickness. The margins of the tumours were clearly demarcated, lobulated, expanding both inner and outer tables and of a homogeneous density (Fig. 1). *Sternum:* This was generally expanded, measuring 3·5 to 4 centimetres in

FIG. 1
Radiograph of skull.

FIG. 2

FIG. 3

Figure 2—Radiograph of sternal mass (necropsy specimen). The general nodularity and spicular structure are well seen. Figure 3—Thoracic spine.

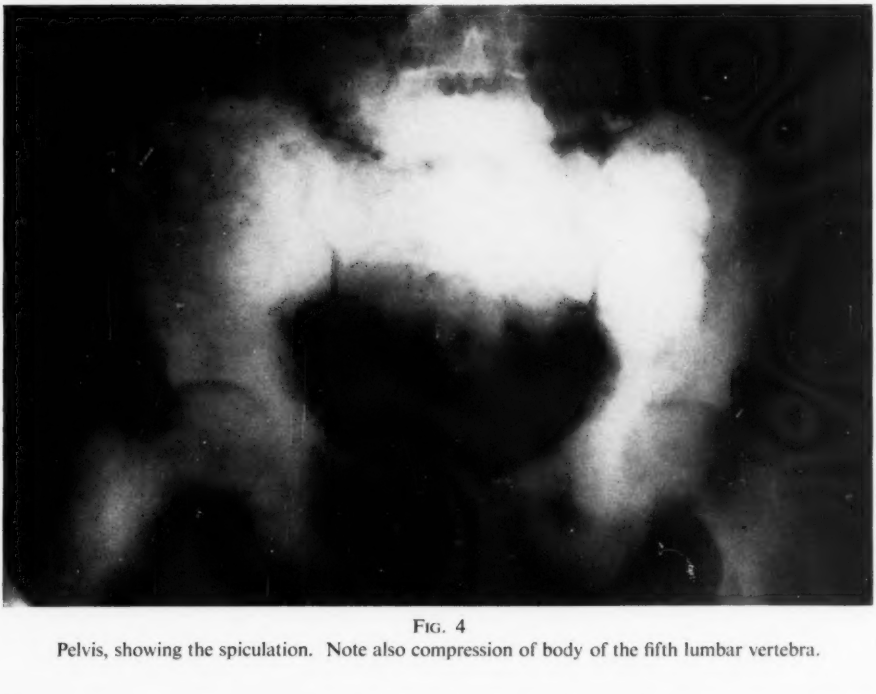

FIG. 4
Pelvis, showing the spiculation. Note also compression of body of the fifth lumbar vertebra.

thickness. The margin posteriorly was clearly defined, but anteriorly was not quite so clear and gave the impression of spiculation on remote viewing (Fig. 2). *Ribs:* The right first rib showed a similar but rather more granular appearance. *Right scapula:* The glenoid fossa and coracoid process were similarly involved. *Right humerus:* The humeral head was dense, showing changes like those of the skull, but with an abrupt distal limit 10·8 centimetres from the upper border of the head of the bone. *Vertebrae:* The bodies of thoracic vertebrae 3–6 and 9–11, and of lumbar vertebrae 2, 3 and 5, had a normal contour, but showed a mottled increase in density. An amorphous mass of calcium density extended into the soft tissues in the region of L.2 and L.5 (Fig. 4). The sacrum was extensively involved (Fig. 4). *Pelvis:* The anatomical form was distorted and the individual bones were thickened by numerous dense confluent masses which in a good film showed a spicular calcified structure. There were few small outlying calcified nodules (Fig. 4). *Femora:* the uppermost third of the shaft, the neck and the head of the right femur were affected, showing again the distal abrupt termination of the lesion as noted in the right humerus. Ill defined diffuse opacities were observed in the lower end of the left femur, but cortex and medulla were still distinguishable (Figs. 5 and 6). *Remaining bones:* Apart from osteoporosis of the long bones, these were normal. *Barium meal and swallow* showed no abnormality. *Intravenous pyelography:* Concentration of the medium was consistently poor in both kidneys, but no other abnormality was revealed.
Investigations—The Wassermann and Kahn reactions were negative. The results of blood examinations are shown in Table I.
Progress and treatment—A biopsy of the superficial and deep parts of the hard sternal mass and adjacent soft tissues showed the features described in detail below, but in the light of the unusual clinical and radiological findings it was then thought that the precise diagnosis was in doubt. The histology was then interpreted as osteogenic sarcoma or secondary carcinomatosis of an extreme osteoblastic type from an undisclosed primary tumour elsewhere.

The patient's steadily increasing discomfort, weakness and pain rendered treatment imperative, but after full consideration both surgery and formal radiotherapy were discarded. It was thought, especially in view of the multiplicity of the lesions, that the condition might respond favourably to large doses of P^{32} administered under close haematological supervision. Hence, from February 22 until May 26, 1951, the bedridden patient was treated with injections of P^{32}, given in five four-weekly doses to a total of 85 μc.

There followed considerable subjective and objective improvement, the patient becoming able once more to dress herself, to stand and to walk unassisted. The several tumour masses, formerly growing, now ceased to enlarge, and those on the skull showed slight but definite regression. Moreover, serial radiographs showed no further advance in the extent of the skeletal changes. However, in early June, while she was out of hospital, her condition deteriorated rapidly within a few days. She became profoundly anaemic, with mucosal bleeding and eventual coma. She was readmitted to hospital and died on June 30, 1951.

The clinical improvement which accompanied the marked fall in the formerly very high level of the serum alkaline phosphatase took place while treatment was being given with radio-phosphorus (Fig. 7).

FIG. 5

Upper end of right femur. Note the uneven density of the tumour with sharp distal delimitation. The horizontal lines indicate the level of the transverse section shown in Figure 6.

Necropsy—This was performed twenty-four hours after death by Dr A. L. Taylor. *Thorax:* The sternum was grossly thickened, its body being expanded by an irregular growth of ivory hardness. Both lungs were congested and oedematous, with bilateral serous pleural effusions. There was no sign of any pulmonary neoplasm, for which a careful search was made. The myocardium showed toxic changes, and there was moderate coronary atheroma. There was also a small pericardial effusion. *Abdomen:* The alimentary and urogenital systems revealed nothing of note. *Lymphoid tissue:* No lymph nodes were involved. The spleen was congested and fibrous. *Endocrine glands:* No macroscopic abnormality was noted. *Skeleton:* In addition to the sternal mass, there were irregular solid outgrowths of the following bones: right parietal, occipital, right first rib, thoracic and lumbar vertebrae, sacrum, both innominate bones, right humerus and both femora. These multiple tumours were well defined and circumscribed, compressing rather than invading adjacent tissues. The masses were all solid, in places rubbery and yielding to the knife, in others densely hard and mineralised. Their colour was uniformly yellowish-white.

Histological examination. *The focal lesion*—The tumour tissue generally showed a characteristic growth pattern. In the individual nodule, lying within a pseudo-capsule of compressed muscle and fibrous tissue, there was a peripheral cambial layer of fairly uniform cells, spheroidal or

FIG. 6

Radiograph of transverse section of femur diaphysis. (×2.) Note the spicular nature of the tumorous outgrowth and the solid intramedullary plug. There is also invasion of the osteoporotic femur cortex.

fusiform in shape (Fig. 8). In certain places these had aggregated into small groups or formed a marginal layer around osteoidal islets. Elsewhere, where the tissue appeared more mature, such cells were absent, and the external fibrous tissue assumed the appearance and some of the functions of a periosteum with foci of lacunar resorption. Deep to this superficial cellular layer were sheets and irregular masses of osteochondroid matrix, which in more centrally placed parts tended to split up into a trabeculated pattern. Although in the ordinary visual microscope this matrix appeared fairly uniform, on examination with crossed polars the pattern of its fibrous structure was quite unlike that of normal osteoid, cartilage or bone (Fig. 10). The superficial cellular zone was not markedly vascular.

More deeply, there was further matrix condensation, with either a trabeculated or reticulated pattern of mineralisation, and fewer tumour cells (Fig. 9). A very few small areas of granular matrix degeneration were seen. In the deeper matrix also there were tissue spaces of irregular shape, some of which contained tumour cells, whereas others showed

TABLE I

RESULTS OF BLOOD EXAMINATION

Date	Serum phosphatase		Serum calcium, milligrams per cent	Serum inorganic phosphates, milligrams per cent	R.B.C. Millions per cubic millimetre	Hb. per cent	W.B.C. per cubic millimetre
	Alkaline. K.–A. units per 100 cubic centimetres	Acid. Bodanski units per 100 cubic centimetres					
19.12.50					4·1	80	8,800 Diffl., normal
27.12.50	128	1·9					
11.1.51			11·7	2·8			
23.1.51			8·2	3·1			
7·2·51	1,500						
10.2.51	1,200	2·7					
2.3.51	720	2·0					
9.3.51	930	4·1					
16.3.51	644				4·0	78	3,600
27.3.51	509					72	5,800
6.4.51	547					94	9,200
19.4.51	456						
5.5.51	300					70	4,000
24.5.51	287						
20.6.51					0·9	16	600 (Marked leucopenia)

normal fibro-areolar tissue together with blood and lymph vessels and a few osteoclasts. The essential tumour cell growth appeared to be by proliferation of spheroidal cells which closely resembled osteoblasts, although in some sites these cells were atypical and somewhat pleomorphic. Mitoses were extremely scanty. Invasion of adjacent muscle by these cells was noted in relation to the sternal mass and the outgrowth of the upper end of the right femur (Fig. 11). No large soft-tissue tumour was found anywhere, such as is usually found in osteogenic sarcoma of Grades II and III.

These features were uniformly seen in the various tumour sections examined. In the proximal long bones (humerus and femur), in addition to the extra-osseous growth, there

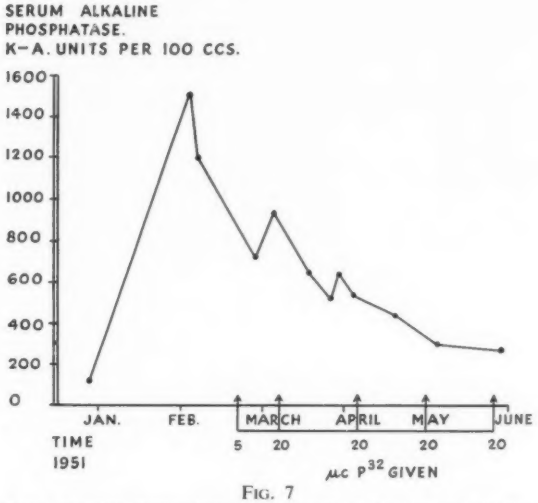

FIG. 7

Curve showing fluctuations of the level of the serum alkaline phosphatase. (The arrows indicate the time of injection of indicated dose of P^{32}.)

was also longitudinal intramedullary extension of tumour tissue, the growing edge being sharply delimited from the adjacent fatty marrow. In such sites there was no clear line of demarcation between tumour tissue and the old mature cortex. Furthermore, in the compact cortex were numerous islets of tumour tissue, lying in the enlarged Haversian canals, which merged almost imperceptibly with the old lamellar bone (Fig. 12). These areas also showed proliferation of the osteoblast-like cells, with the formation of much new osteoid. (This mode of cortical invasion is typical of the less aggressive forms of osteoblastic osteogenic sarcoma in which matrix formation is relatively more advanced than tumour cell proliferation. Thus invasion of the Haversian systems of the cortex is extensive, with little or no bone destruction. In fact, in many places the bone texture is over-dense as a result of the intracortical deposition of tumour osteoid.)

Non-tumorous bone—A number of blocks were examined. Age changes were noted in lamellar bone, together with fairly marked osteoporosis, this being most pronounced in the femur diaphysis where the cortex was reduced to a thin shell. An extensive search for evidence of Paget's disease was negative.

Skin (from chest)—This was atrophic, with absence of the normal dermal papillae. Moderate hyperkeratosis was present, with horny follicular plugs. The cells of the stratum germinativum showed increased brown pigment, and a few scattered chromatophores were seen in the relatively acellular sclerosed dermis.

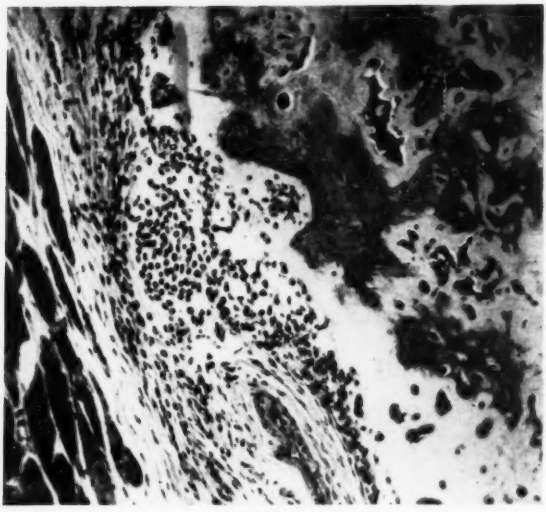

FIG. 8

Photomicrograph. Growing edge of sternal mass. This field shows the superficial cellular layer with here a relatively narrow zone of unmineralised osteochondroid matrix. The more darkly stained material is bone. Elsewhere the unmineralised zone was of great depth. (Haematoxylin, Phloxin and Tartrazine, ×120.)

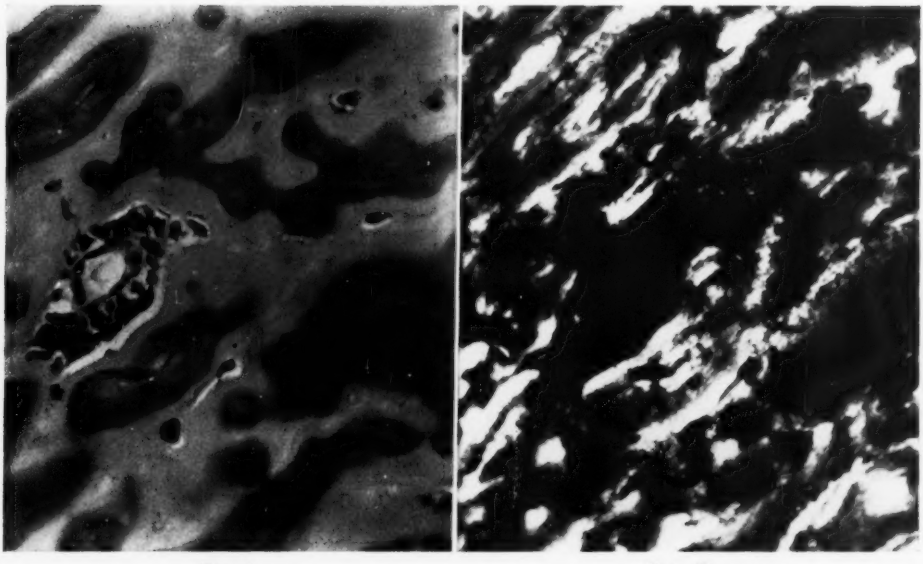

FIG. 9 FIG. 10

Figure 9—Deeper region of a small nodule from inner surface of parietal bone. Shows a small group of tumour cells embedded in the relatively acellular matrix, which is partly bony. Figure 10—Same field as Figure 9, shown through crossed polars. Note unusual pattern of fibrous stroma of matrix. (Haematoxylin, Phloxin and Tartrazine, ×240.)

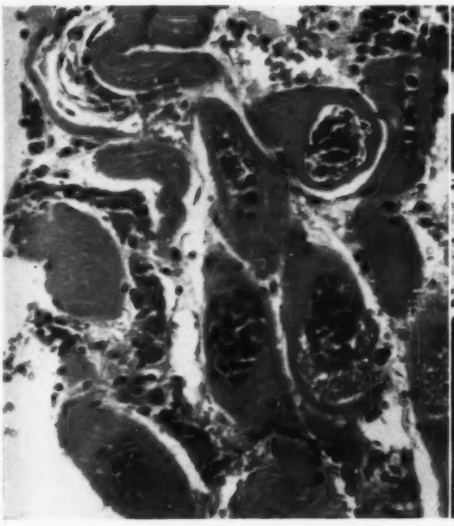

FIG. 11

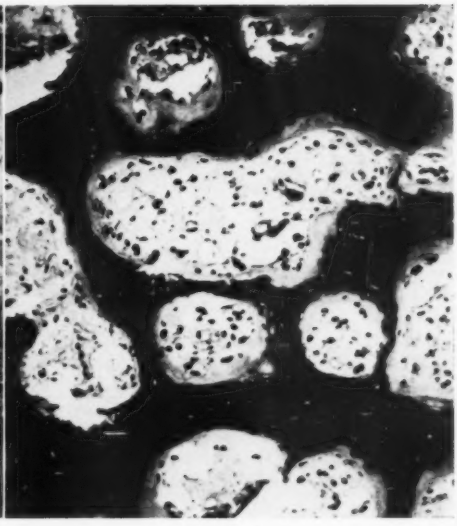

FIG. 12

Figure 11—Edge of sternal nodule. Note destructive invasion of muscle fibres by typical spheroidal tumour cells. (Haematoxylin and Eosin, ×225.) Figure 12—Transverse section of femoral shaft from region indicated by lines in Figure 5, and shown in full in Figure 6. Note invasion of the grossly enlarged central canals of the Haversian systems. In this tubular form of growth the active cellular layer is central, not peripheral. (Haematoxylin, Phloxin and Tartrazine, ×120.)

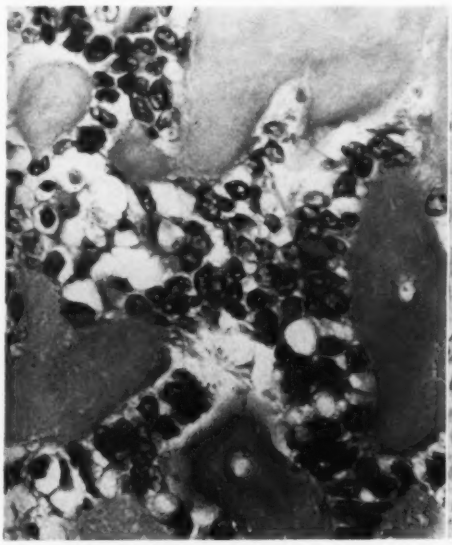

FIG. 13

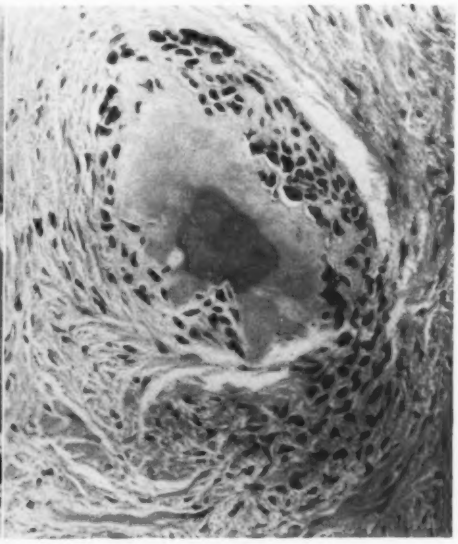

FIG. 14

Figure 13—Nodule on inner surface of parietal bone. The cellular superficial parts of the tumour masses at all sites shows this pattern of spheroidal or angular cells closely related to irregularly trabeculated osteoid. (Haematoxylin and Eosin, ×335.) Figure 14—Outlying tumour nodule near the edge of the sternal mass. Note the peripheral cambial layer and central areas of uncalcified and calcified osteoid. (Haematoxylin, Phloxin and Tartrazine, ×215.)

Abdominal viscera—The liver showed diffuse fatty degeneration, with accumulation of amorphous material in the sinusoids. The kidneys and pancreas were normal.

Endocrine glands—The pituitary weighed 0·65 gramme. There was no naked-eye or microscopic abnormality. Four parathyroids were found in the normal cervical position. Total weight (wet) was 0·206 gramme. On section, most of the cells were of the clear type, with very few oxyphil cells throughout. The thyroid weighed 30 grammes. Microscopically it showed normal vesicular structure with ample colloid storage and no interstitial fibrosis. A few lymphorrhages were seen. The suprarenals appeared normal, and the ovaries were normal for the age.

COMMENT

To the histologist acquainted with the cell morphology and growth pattern of low grade osteoblastic osteogenic sarcoma, there can be no doubt that this present case is an example of the less actively growing type of this tumour. In the Bristol Bone Tumour Register collection there are three similar but unifocal tumours; these have arisen in patients aged twenty-one, thirty-one and forty-seven years, and have been primary in rib (2) and navicular of foot. Another similar growth is that reported by King (1932). In this form of osteogenic sarcoma slow growth and absence of the otherwise common early pulmonary metastases are typical, although local recurrence may follow incomplete surgical removal. In the multiple lesions here reported the histological features closely resemble those seen in the rib tumours mentioned above. A similarity may also be sometimes observed in osteogenic sarcoma of Grade II, but here, and even more markedly in Grade III tumours, the rapid tumour cell proliferation quickly obliterates all tissue landmarks, thus rendering the structure chaotic and diverse.

The general behaviour of tumours of this type, when solitary, favours a good prognosis on account of the relative infrequency of early pulmonary involvement. Although in our patient the clear lung fields argued strongly against former haemic spread from any one osseous site, this possibility is not completely excluded. Nevertheless the clinical, radiological and histo-pathological findings are more suggestive of multiple primary tumours than of a single sarcoma (possibly of the sternum) with numerous blood-borne osseous metastases.

While we incline to the view that the lesions represent multiple primary tumours, it is necessary to bear in mind that it was only in relation to the sternal and femoral masses that invasion of adjacent tissues was observed. In sections taken from many skeletal sites the histology, although atypical, did not suggest active aggressive neoplasia. It may well be that the correct interpretation of the condition is that of a widespread osteoblastic bone dystrophy, in which sarcoma has supervened simultaneously at several points. There may be a background factor in the osteoporosis, which in the femur seemed excessive for a woman of fifty years. This bone change is probably post-menopausal in origin with the additional effect of disuse atrophy in the lower limb. Certainly in persons over the age of fifty years at least half of all examples of osteogenic sarcoma have concomitant Paget's disease (Price 1955). In a number of osteogenic sarcomata in patients of this age, but without Paget's disease, well advanced osteoporosis has been present with extensive bone disorganisation which in certain ways is akin to the osteitic process.

The multifocal nature of about a fifth of all bone sarcomata complicating Paget's disease is well recognised. Paget's sarcoma is usually very cellular, markedly osteolytic and rapidly fatal; cell proliferation is rapid and active; so connective tissue differentiation in the tumour proceeds beyond the fibroblastic stage in relatively few instances. In the present case no evidence was found of Paget's disease, nor does the behaviour and structure of the tumour conform with any of the variants so far observed in twenty-five personally studied sarcomata associated with osteitis deformans.

In the case described by Silverman (1936) the multiple lesions noted in the pleurae and solitary nodules of the pericardium and colonic peritoneum strongly suggested a metastatic

origin, although it is conceivable that even these extraskeletal foci were in fact further primary lesions. This explanation would be consistent with the concept of multicentric neoplasia superimposed upon a systematised mesenchymal dystrophy. Silverman's observation in her material that there was marrow replacement by bone-forming fibrous tissue beyond the limits of definite sarcomatous invasion strengthens this view. Furthermore, extraskeletal bone-forming and cartilage-forming tumours are well known, and are periodically reported, most often in muscle, breast, thyroid and urinary bladder.

Lichtenstein (1952) mentioned a patient of Dr G. L. Kraft who had a multicentric osteogenic sarcoma. No details are cited, but the radiographs reproduced, particularly that of the pelvis, closely resemble those of our patient reported here.

The literature has recently been reviewed by Finlayson (1953), with the exception of the case reported by Moseley and Bass (1956). These latter authors comment upon the similarity of the radiological features in the published cases, with which broadly the present findings agree. In the case mentioned here, and in that reported by Busso and Schajowicz (1945–46), pulmonary metastases were absent.

Apart from the clinical evidence of some ill-defined endocrine dysfunction there were no other features of possible etiological significance in the present case.

SUMMARY

1. The detailed findings are presented of a woman aged fifty who had widely distributed ossifying skeletal tumours, the structure and form of which have been shown to be low-grade osteoblastic osteogenic sarcoma. The patient was treated with heavy doses of P^{32}, to which is attributed some clinical improvement, but which induced a fatal aplastic anaemia.

2. The study of the several tumours, together with the history, the radiographs and the post-mortem findings, suggests that the lesions are multifocal skeletal primary tumours rather than numerous osseous metastases secondary to a solitary new growth in any one bone.

3. The focal neoplastic lesion is discussed in the light of personal experience of other osteogenic sarcomata of low grade but of solitary origin, and of the multifocal form of bone sarcoma which may complicate Paget's osteitis deformans.

Our thanks are due to Dr R. C. Tudway, Director of the Radiotherapy Department of the Bristol Royal Hospital, for permission to publish this case, and to Dr A. L. Taylor, Pathologist of the same institution, for the gift of necropsy specimens and access to his records.

This case has been reviewed in detail as part of a wider study of the large series of bone sarcomata compiled by the Bristol Bone Tumour Register, which is supported by the British Empire Cancer campaign.

The reproductions of radiographs and photomicrographs are the work of Mr J. E. Hancock.

REFERENCES

BUSSO, M. G., and SCHAJOWICZ, F. (1945–46): Sarcoma osteogénico a localización multiple. Revista de Ortopedia y Traumatologia, **15**, 85.

FINLAYSON, R. (1953): Osteogenic Sarcoma with Multiple Skeletal Tumours. Journal of Pathology and Bacteriology, **66**, 223.

KING, E. S. J. (1931): An Example of Benign Osteogenic Sarcoma. British Journal of Surgery, **19**, 330.

LICHTENSTEIN, L. (1952): Bone Tumours, p. 171. London: Henry Kimpton.

MOSELEY, J. E., and BASS, M. H. (1956): Sclerosing Osteogenic Sarcomatosis. Radiology, **66**, 41.

PRICE, C. H. G. (1955): Osteogenic Sarcoma; an Analysis of the Age and Sex Incidence. British Journal of Cancer, **9**, 558.

SILVERMAN, G. (1936): Multiple Osteogenic Sarcoma. Archives of Pathology, **21**, 88.

AIR EMBOLISM AS A COMPLICATION OF MEDULLARY NAILING

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The satisfactory results obtained from medullary nailing are well known, and the method is widely used in fracture work. It is thus important to call attention to yet another danger inherent in the technique—namely air embolism. Simple means of overcoming this danger are also referred to.

CASE REPORT

A man of thirty-one sustained a minor head injury with transient loss of consciousness, a closed fracture of the right tibia and fibula, and a closed fracture of the mid-shaft of the right femur. His general condition presented no problem and he recovered rapidly from the head injury. The fractured tibia and fibula were immobilised in plaster. There was gross displacement of the fractured mid-shaft of femur, uncorrectible by manipulation, and it was obvious that there was muscle interposition. Operative reduction and medullary nailing were undertaken through an antero-lateral incision. The patient was thin, and all vessels were picked up before they were divided, the fracture thus being exposed with practically no bleeding. At that time the practice in this hospital was to determine the calibre of the nail to be used by trial and error. The narrowest part of the medullary cavity being in this case above the fracture, a nail was passed along the marrow cavity for six or eight inches to test for size. The first nail tried was obviously too loose and the second was still a little loose, but it was decided that the third nail would fit snugly. This nail was then withdrawn and a guide wire was passed up the bone from the fracture site and out through the greater trochanter. A small incision was made to expose the point of the guide wire, and the nail was hammered over it. A perfect reduction was obtained and the operation was completed by hammering the nail into the lower fragment. The operation lasted no more than forty-five minutes and the patient's condition gave no cause for anxiety, but as he was being lifted from the operation table he collapsed. Respiration was maintained but no heart sounds could be heard. Attempts at resuscitation by cardiac massage were carried out and an intra-cardiac injection of adrenalin was given, but without avail.

Post-mortem examination—There was some muco-pus in the lower end of the trachea and in the main bronchi. The lungs were a little congested, with evidence of early pneumonia in the left lower lobe. The right side of the heart contained a large quantity of frothy blood, particularly the right ventricle. There was no visible coronary atheroma, and the heart was normal in other respects. The abdominal organs were normal, as were the skull, brain and meninges. When the medullary nail was removed from the femur the medullary cavity was found to be virtually devoid of marrow, resembling a length of empty pipe.

DISCUSSION

It is clear that the cause of death in this case was air embolism. The history of sudden collapse in a previously satisfactory patient, the continuation of respiration coupled with absence of heart sounds, the ineffectiveness of cardiac massage, and finally the finding at necropsy of a large quantity of frothy blood in the heart, will admit of no other explanation. It may be argued that the early left lower lobar pneumonia contributed to the death, but it is inconceivable that it was other than a minor factor in view of the sequence of events.

The question arises—what was the port of entry of air into the circulation? No vessels of any size had been divided before clamping. Indeed the whole operation was remarkably free from bleeding, and at necropsy no gaping or unsutured vessels were found at the site of operation. One of us, who performed the necropsy (M. A. L.), was impressed by the empty pipe-like appearance of the femur, and formed the opinion that the only possible site of air entry was through the marrow, which is very vascular. At the time of necropsy it was not known that more than one pin had been inserted along the medullary cavity. Subsequent discussion with the operator (A. H. S.) brought to light the practice of nailing by trial and error, as referred to in the case history. It is clear that if more than one nail is passed along the marrow cavity the action of the first nail will be to clear out much of the marrow and fat, so converting the marrow into a hollow tube which will immediately fill with air when the nail is withdrawn. Subsequent nails passed along the cavity will exert a plunger action and pump air into the sinusoids of the remaining marrow which forms a lining to the cortical bone. The groove of the Küntscher nail may easily become occluded by marrow debris, thus perfecting the plunger action. The amount of air thus forced into the circulation will clearly increase with the number of nails that are " tried for fit." At this point one should stress that the practice in this hospital is now to rely on radiological assessment of the calibre of the medullary cavity.

Reference to the literature has failed to reveal any report of a similar case. But air embolism during antral irrigation is well recognised, and the mode of entrance of air into the circulation may be similar to that in our case. Thus it is generally accepted that air may enter the circulation through the veins of the antral mucosa (Cornfield and Laszlo 1949), and it may be expected that air may enter the circulation through the equally vascular thin lining of marrow remaining after nailing a fractured femur.

The literature brings out some general points of importance in regard to air embolism that have a bearing on the present case. Fleischmann (1940) referred to the accelerating effect of an upright or sitting posture on air embolism in ear surgery. Conversely, in our case the patient was horizontal during the operation; so there would be some facilitation of venous return in the legs over and above that present in the standing position. This in turn would favour the entry of air into the venous circulation through the marrow sinusoids.

During inspiration the negative intrathoracic pressure is increased, and there is a consequent increase in the negative pressure in the right auricle, which aids the sucking of blood (and air if present) into the chest. Any undue inspiratory efforts are therefore likely to increase the risk of air embolism (Hepler, Truter and Hunt 1947). In the present case, however, there is no evidence of excessive respiratory excursions.

Another bone whose fracture may lead to air embolism is the skull. Air embolism is more likely to occur if the base of the skull is fractured, the fracture being associated with rupture of a sinus and an opening to the exterior. Nevertheless the diploic veins of the skull can themselves form the site of entry of air, and these veins do not usually collapse, because the bone supports them (Roer 1949). The question of posture is again important in regard to air embolism from a fractured skull; its likelihood is obviously diminished by assuming a horizontal posture, because the " suction pressure " is thereby decreased. The importance of posture and of " suction pressure " is also well illustrated by Kovacs (1938), who described a case of air embolism after a hand wound in which elevation of the hand above the heart level continued for some time after infliction of the wound, and this was considered to be an important factor in causing air embolism.

Whenever the question of air embolism is discussed the question of the volume of air necessary to cause death is raised. No simple answer can be given, and much depends on the patient's general condition at the time of embolism. Thus a feeble old man will succumb to the effects of a smaller volume of air in the venous circulation than will a young athlete. The rate of entry of air is obviously of great importance, because air injected slowly will

partly dissolve in the blood and the air that does not dissolve will form relatively small scattered bubbles which may not cause any serious ill effect. On the other hand, the same volume of air injected rapidly may be expected to cause a homogeneous consecutive series of air bubbles which all reach the right heart in rapid succession, and, cumulatively, cause a mass of bloody froth in the comparatively small right auricle and ventricle. Once such a mass of bloody froth is present in the heart, grave impairment of the heart's action can be expected. Such froth does not seem to be passed on readily into the pulmonary circulation, possibly because of spasm of the pulmonary artery such as is believed to occur in ordinary pulmonary embolism. It is notable that Chase (1934) produced arterial constriction in rabbits in the course of experimental air embolism.

Posture and the volume of air sucked into the veins are therefore the two most important factors in the etiology of air embolism. In the present case the horizontal posture, as indicated above, would tend to facilitate entrance of air into the venous circulation. The volume of air in this case was probably considerable, some air being forced into the veins through the marrow on each occasion that the nail was reinserted. It is likely that if the operation had been carried out with the patient in a comparatively " head up " position, instead of the more usual horizontal position, the marrow cavity would have filled with blood rather than air and thus the repeated nailing would have been harmless. In the " head up " position, however, the wound is more likely to be flooded with blood, so obscuring the operative field. An alternative that would enable the patient to be kept horizontal would be to flood the wound and the opening into the marrow with saline to prevent the access of air. Nevertheless there is likely to be less efficient filling of the marrow cavity by saline than by the natural means of bleeding into the wound resulting from a " head up " position (in the case of a wound below the heart).

It seems preferable to rely entirely on radiological assessment of the calibre of the marrow cavity, by strapping a nail to the lateral aspect of the thigh. The correct calibre of nail can then be chosen with precision.

DIAGNOSIS AND TREATMENT

The first essential in the diagnosis of air embolism, upon the rapid establishment of which any hope of effective treatment depends, consists in bearing the possibility in mind whenever there is sudden collapse after a wound, whether accidental or surgical. Cyanosis rapidly becomes evident. With the possibility in mind, the nature of the wound may indicate immediately whether any veins are likely to have been opened, into which air could be sucked. Secondly the position of the wound in relation to the heart and the patient's posture (either the present posture or the posture at the time of infliction of the wound) must be assessed. In the case of large veins, with suction of a large volume of air, it is likely that a hissing sound will be heard and so immediately establish the diagnosis. But hissing is not always present; in its absence, when the possibility of air embolism is realised, the heart should be auscultated. If air embolism has occurred the heart sounds are at first loud and roaring (Leetch 1938). Thereafter the heart soon weakens and stops within a matter of minutes. Respiration continues, and if the heart has not been auscultated soon enough to hear the roaring sound, the combination of absent or weak heart sounds with continued respiration in a suddenly collapsed cyanotic patient with a wound, makes the diagnosis of air embolism virtually certain.

In treatment a pad of any material immediately available must be firmly applied to the wound in an effort to prevent further access of air. At the same time the patient's posture must be immediately altered, if necessary, making the wound dependent, so as to diminish the " suction pressure." The next step consists in attempting to remove air from the heart; all that is needed is to insert a needle into the right ventricle and, with a syringe, to suck out the bloody froth. It may be necessary to continue this for some time, and to combine it with cardiac massage. To enter the right ventricle blindly the needle, which should be 6 to 10

centimetres long, is inserted at the left sternal border at the upper edge of the fifth intercostal cartilage, and directed slightly inwards (Lembcke 1946). But it is preferable to insert the needle under direct vision after exposing the heart by thoracotomy.

SUMMARY

A case of fatal air embolism after Küntscher nailing of a fractured femur is described. Necropsy indicated that the only possible means of air entry was through the bone marrow. Subsequent discussion between the surgeon and the pathologist indicated that air must have been forced into the venous circulation through the marrow by repeated removal and reinsertion of nails, which allowed air to fill the punched-out marrow space when the nail was removed, the same air being forced into the marrow sinusoids when the nail was reinserted and hammered into position. This danger may be overcome 1) by allowing the site of operation to flood with blood by placing the patient in a "head up" position; 2) by flooding the operation site with saline; or 3) by assessing the calibre of nail required by radiological means rather than by trial and error.

Grateful acknowledgment is made to Mr E. A. Nicoll for access to the clinical notes.

REFERENCES

CHASE, W. H. (1934): Anatomical and Experimental Observations on Air Embolism. Surgery, Gynecology and Obstetrics, **59,** 569.

CORNFIELD, E., and LASZLO, A. F. (1949): Fatal Air Embolism During Antral Irrigation. Annals of Otology, Rhinology and Laryngology, **58,** 479.

FLEISCHMANN, L. (1940): Ueber Luftembolie bei Ohroperationen. Practica Oto-Rhino-Laryngologica, **3,** 295.

HEPLER, T. K., TRUTER, J. L., and HUNT, H. F. (1947): Air Embolism Occurring During Mastectomy: Report of a Fatal Case. American Journal of Clinical Pathology, **17,** 322.

KOVACS, H. J. (1938): Über einen Fall von Luftembolie nach einer Verletzung der Hand. Zentralblätt für Chirurgie, **65,** 180.

LEETCH, H. W. (1938): An Unusual Accident Occurring in Closed Intrapleural Pneumolysis. Journal of Thoracic Surgery, **8,** 230.

LEMBCKE, W. (1946): Über erfolgreiche Behandlung einer schweren Luftembolie durch Herzkammerpunktion. Chirurg, **17–18,** 31.

ROER, H. (1949): Luftembolie als akute Gefahr bei Unfällen. Medizinische Klinik, **44,** 1,157.

REPORT OF THE TREATMENT OF EXPERIMENTAL FAT EMBOLISM WITH HEPARIN

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The clinical manifestations of fat embolism may be seen quite frequently by those responsible for the treatment of the injured, and the condition is diagnosed even more commonly by pathologists making special search for it in persons who have died after injury (Robb-Smith 1941). As Harris (1955) has recently stressed, many problems concerning the source of the fat and the cause of its appearance remain undecided, and the surgeon is frustrated by the lack of any specific prophylactic or therapeutic measure. Hahn (1943) reported that heparin had the effect *in vivo* of "clearing" lipaemic plasma—that is, of rendering plasma which had become milky and opalescent after a fatty meal again translucent, and it seemed possible that heparin might thus be of value in the treatment of fat embolism. If the chylomicra of lipaemic plasma are so effectively lysed (French, Robinson and Florey 1953), might not the fat particles present in the blood in fat embolism be similarly removed, thereby decreasing the risk of continuing or repeated visceral infarction? This was the rationale for a study of the effects of heparin in experimental fat embolism.

The results of such an investigation are presented in this report, and they suggest that a clinical trial of heparin in human fat embolism is not only unwarranted, but might be dangerous.

MATERIALS AND METHODS

Witzar strain rats were used for the experiments, and embolism was produced by injecting the fat into the femoral vein exposed at the groin under Nembutal anaesthesia. Each injection was given evenly during one minute.

With the object of simulating as closely as possible naturally occurring fat embolism, homogenous fat was used: it was prepared as follows.

The perirenal and epididymal fat pads of freshly killed rats were stored in absolute ethyl alcohol; anaesthetic ether (2 pints to 1 of alcohol) was then added and the tissue was minced up in a Waring blender. After filtration the alcohol and ether were evaporated off under reduced pressure, the fat was brought up to 100 degrees Centigrade to coagulate any tissue protein still present, and it was filtered again and set up in glass ampoules. These were sealed after autoclaving for fifteen minutes at five pounds per square inch.

The minimal lethal dose (M.L.D.) of the fat varied slightly from batch to batch and the dosage used in the experiments was accordingly varied with each batch to keep the fatality rate fairly constant.

In the first series of experiments reported below heparin ("Liquemin" brand (Roche) 5,000 i.u./millilitre) was injected into the femoral vein in a dose of approximately 0·6 milligrams per 100 grammes body weight either immediately before or immediately after the fat had been given. In the second series heparin was administered by the combined intraperitoneal (0·6 milligrams per 100 grammes) and intramuscular routes (Heparin Retard (Boots) 1·2 milligrams per 100 grammes). Clotting times were estimated to confirm that the intraperitoneal heparin had been absorbed.

The experiments were controlled as follows. Fat embolism was induced in a series of rats, but heparin was given to only half of the animals. The time elapsing before death, if this occurred, was recorded and a post-mortem examination was performed on all animals that died. The groups of figures depicting survival times, presented below, were each compiled from a series of such experiments, each fully controlled, but not performed on the same occasion.

RESULTS

Fat embolism—In suitable dosage the intravenous administration of homogenous fat by the technique described above usually caused death one to twelve hours after the injection of the fat. Deaths occurring instantaneously or more than thirty-six hours after injection were rare; the former were ascribed to shock, the latter were often associated with clot retention in the bladder from haematuria. It was not uncommon to detect signs highly suggestive of cerebral fat embolism (ocular palsies, ataxia, fits, hemiparesis) in animals surviving for more than a few hours after injection. Petechial haemorrhages were common, particularly in the thymus of animals dying late.

Post-mortem examination showed dilation of the right side of the heart and secondary pleural effusions. Microscopically the intrapulmonary vessels were seen to be laden with fat and it seemed likely that death was due to anoxia following right heart failure.

The effect of heparin on fat embolism—The effects of administering heparin together with an intravenous dose of 0·16 millilitre fat per 100 grammes body weight are shown in Figure 1 and Table I. Table II shows the results in a further similar series, also in the form of survival times. From these it will be seen that heparin greatly accelerates the mortality from fat embolism in the first four hours after its induction, and also increases the total mortality over an arbitrary observation period of forty-eight hours. Heparin alone, in three times the doses used in these experiments, had no lethal effect on ten rats which were given 0·1 millilitre intravenously.

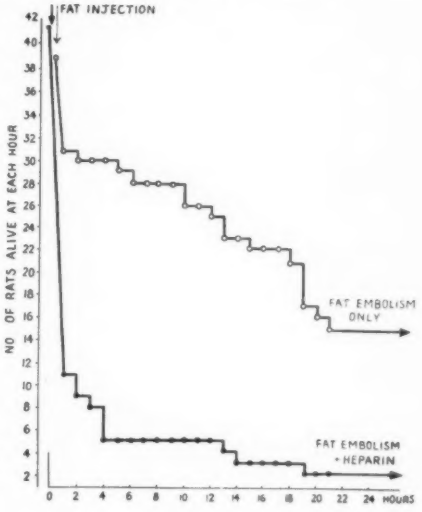

FIG. 1

Graph showing the effects of heparin in experimental fat embolism (solid circles) compared with a control group of animals which suffered fat embolism alone (clear circles). Details of dosage of fat and heparin as stated in text.

There were no macroscopic or microscopic differences observed at post-mortem examination between the animals that died after fat embolism alone and those that had also received heparin. More particularly, we were unable to detect haemorrhages in viscera or tissue spaces from giving heparin to the latter group of animals.

DISCUSSION

It has been shown that the administration of heparin to rats in which homogenous fat embolism has been experimentally induced causes a great acceleration of mortality in the early stages after the initial embolism and also increases the ultimate mortality. Post-mortem examination of the cadavera of the rats and microscopy of their tissues reveals that the deaths promoted by heparin are not due to internal haemorrhage, which theoretically might have been caused by the best known of the properties of heparin, the anti-coagulant effect.

It is certain that many features of the fat embolism induced in these experiments differ from those that obtain in man after injury. Nevertheless it seems likely that heparin would also be harmful in human fat embolism, and this report is occasioned partly by this conclusion, lest others, reading the papers that aroused our interest in the subject, might be tempted to

TABLE I

TIME-TABLE OF DEATHS IN RATS SUFFERING EXPERIMENTAL FAT EMBOLISM. Fat dosage 0·16 millilitres per 100 grammes body weight. This shows that heparin both accelerates death and increases the number of deaths.

	Deaths	
Time	Fat embolism alone	Fat embolism plus heparin
0 to 2 hours	9	32
2+ to 12 hours	5	4
12+ to 48 hours	10	3
Number of rats surviving .	15	2
Total number of rats . .	39	41
Percentage of rats surviving	38 per cent	5 per cent

try the effect of the drug on such patients. At the outset of this work we felt that heparin therapy in human fat embolism, apart from the controllable risks of haemorrhage, could at the very worst " do no harm "; it now appears that this premise may be incorrect.

Further experiments not detailed in this report have shown that this effect of heparin is not limited to fat embolism, but is similarly exerted in the presence of emboli of entirely

TABLE II

DEATH TIME-TABLE IN AN EXPERIMENT WITH RATS LIKE THAT IN TABLE I, except that heparin was given both intraperitoneally and intramuscularly. Fat dosage 0·18 millilitres per 100 grammes body weight.

	Deaths	
Time	Fat embolism alone	Fat embolism plus heparin
0 to 2 hours	4	13
2+ to 12 hours	13	17
12+ to 48 hours	11	1
Number of rats surviving .	9	4
Total number of rats . .	37	35
Percentage of rats surviving	24 per cent	11 per cent

different physico-chemical nature. These experiments with heparin and embolisms with other oils and gases, together with electro-phoretic studies, indicated that the action of heparin was on the protein at the fat-plasma or the oil-plasma interface: this will be reported in detail by one of us (A. M. N. G.) elsewhere.

SUMMARY

1. Reports of the lipaemia-clearing effect of heparin suggested that this drug might be of therapeutic value in fat embolism.
2. In an experimental trial with rats, heparin was found both to accelerate death and to increase the number of deaths after fat embolism.
3. It is accordingly concluded that a clinical trial of heparin in human fat embolism would not be warranted.

We would like to express our thanks to Professor J. Trueta and Dr A. H. T. Robb-Smith for their guidance and for defraying the expenses of this investigation, and to Dr R. Sladden for his assistance in the microscopic examination of the post-mortem material. The help given by Mr W. Charles in the performance of the experiments was invaluable.

REFERENCES

FRENCH, J. E., ROBINSON, D. S., and FLOREY, H. W. (1953): The Effect of the Intravenous Injection of Heparin on the Interaction of Chyle and Plasma in the Rat. Quarterly Journal of Experimental Physiology, **38**, 101.
HAHN, P. F. (1943): Abolishment of Alimentary lipemia Following Injection of Heparin. Science, **98**, 19.
HARRIS, R. I. (1955): Personal communication.
ROBB-SMITH, A. H. T. (1941): Pulmonary Fat-embolism. Lancet, **i**, 135.

LOCAL GIGANTISM

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Local gigantism is uncommon. Kanavel (1932) asserted that less than seventy-five cases of megalodactyly had then been reported. Referring to this condition Elkeles (1951) stated: " Recording of rare congenital disorders is not only justified on scientific grounds, but their study and grouping may throw light on problems of physiology and on the etiology of diseases."

CASE REPORT

The patient was a European girl of two and a half years. There was no family history of congenital deformity. She was an only child. Her past illnesses included an attack of

FIG. 1
Hands at the age of four and a half years.

cerebral malaria at the age of thirteen months, a second attack of malaria at the age of seventeen months, and an attack of acute tonsillitis at the age of sixteen months. She had otherwise been well.

When the child was two and a half years old her mother first noticed, while bathing her, that she had enlargement of her left thumb and right little finger. She had apparently suffered no pain and had not been feverish. Examination revealed diffuse fusiform swelling of the left thumb in the region of the interphalangeal joint, and of the right little finger at the level of the proximal interphalangeal joint (Figs. 1 and 2). There was no reddening of the skin or increased heat over the affected parts. There was no tenderness or limitation of movement. There was no affection of any other bone or joint.

Further examination revealed no abnormality of heart, lungs, abdomen or nervous system. No nodules were found in skin or subcutaneous tissue. No areas of pigmentation

were seen on the skin. There was no thickening of any peripheral nerves. There was no enlargement of spleen, liver or lymphatic glands. Her teeth and tonsils appeared healthy. The child had a healthy general physical appearance.

Investigations—Leucocyte count showed total leucocytes 13,500 per cubic millimetre (neutrophils 42 per cent, lymphocytes 58 per cent). No abnormality of the cells was seen. Urine examination revealed no abnormality. The Kahn test was negative. The Mantoux test (1 in 1,000) was negative. The erythrocyte sedimentation rate was 5 millimetres in 1 hour (Westergren method).

Progress—The child was observed at intervals for three and a half years. At the age of five and a half years enlargement of the right second and third toes was observed. The left ring finger had also become slightly swollen. Examination six months later showed enlargement of the left thumb, left ring finger, right little finger and right second and third toes. The left thumb and right little finger had remained almost stationary over the past year, though measurements suggested that the thumb had decreased very slightly in circumference.

FIG. 2
Photograph showing marked enlargement of the left thumb.

Radiographic examination (Figs. 3 and 4)—The proximal and middle phalanges of the right little finger, the metacarpal, proximal and distal phalanges of the left thumb, and the proximal and middle phalanges of the left ring finger showed hypertrophy of their diaphyses without evidence of a disease process. Similar but slighter changes were seen in the right second and third toes. The appearances suggested congenital local hyperplasia (local gigantism) of a mild type. Radiographs of the lungs revealed no abnormality.

DISCUSSION

The etiology of local gigantism is obscure. The deformity has been congenital in several of the reported cases (two cases described by Elkeles (1951), four cases of Moore (1942), one case of Inglis (1950), one case of Capel (1935) and one case of Kaplan (1947)), but Capel (1935) put forward the following arguments against its being a purely genetic condition: " In only one of the reported cases is there a family history of the condition: in almost every case it is unilateral, and only rarely is it associated with other congenital abnormalities. The other congenital abnormalities of the fingers which do give clear evidence of inheritance are usually bilateral, and they are very frequently associated with other congenital defects." Cases of

FIG. 3
Radiographs of hands.

FIG. 4
Radiographs of feet.

bilateral but asymmetrical macrodactyly have, however, been reported by Killinger (quoted by Kanavel 1932), by Mouriquand and Buche (quoted by Kanavel 1932) and by Capel (1935). Moore (1942) described bilateral local gigantism of toes. The case reported here shows a combination of local gigantism of both hands with a similar condition of one foot. The lack of symmetry between the two sides in most of the recorded bilateral cases is of significance.

As Moore (1941) pointed out, local gigantism is unlikely to result from the action of a hormone, " which by its very nature must have access to all parts of the body."

Weber (1918, 1951) divided local hypertrophy into two categories: 1) true gigantism, in which the blood vessels are not more involved than the other tissues of the hypertrophied part; and 2) angiectatic hypertrophy. Wright (1951) considered that haemangiomatous and lymphangiomatous conditions have much in common, and that it is possible that they both cause alteration in the size of the developing limb, sometimes an increase, sometimes a decrease. One of Elkeles's (1951) patients displayed a lymphangiomatous condition of the right palm in association with local gigantism of the right second and third fingers.

Evidence strongly suggests that macrodactyly is related to neurofibromatosis (Inglis 1950). Brooks and Lehman (1924) stated: " We know of no other condition in which there is a spontaneous excessive growth in length of a single bone other than Von Recklinghausen's disease." The presence of neurofibromatosis may, however, be overlooked in children, for Moore (1942) noted that cutaneous tumours seldom occur in children, even when typical pigmentary changes are present; but he stated that examination of the finer terminal branches in cases of proved neurofibromatosis showed a pathological picture strikingly like that in the sections from cases of macrodactyly. Cases of local gigantism associated with neurofibromatosis are recorded by Moore (1941, 1942), Bell and Inglis (1925), Baraldi and Ruiz (1934) and Brooks and Lehman (1924), who give references to other cases. Cases associated with areas of pigmentation, but without cutaneous tumours, are cited by Elkeles (1951), Moore (1942), Adair, Pack and Farrior (1932) and Kaplan (1947). A family history of Von Recklinghausen's disease, which was regarded by Moore (1942) as collateral evidence since it is frequently hereditary, was recorded by him in one of his cases of local gigantism. Other conditions that have been shown by Adair *et al.* (1932) to have close clinical analogy to neurofibromatosis and which have been found in cases of local gigantism are multiple lipomata (Inglis 1950, Geunther quoted by Zondek 1944, Moore 1942 and Adair *et al.* 1932) and angiomata (Moore 1942, Elkeles 1951).

As Moore (1941) pointed out, the strongest argument in favour of the view that the neurofibroma is the cause of the hypertrophy is the fact that there is a definite segmental relationship between the affected nerve and the overgrowth, as was well demonstrated by his own case of marked fusiform enlargement of the right median nerve associated with gigantism of the right two middle fingers, and by that of Bell and Inglis (1925) of plexiform neuroma of the median nerve leading to the enlarged part. A similar segmental relationship between neurofibromatosis and splanchnomegaly has been observed by Winestine (1924), who recorded a case presenting papillary adenomatosis of the rectum combined with neurofibromatosis of the pelvic and mesenteric plexus, and who referred to accounts by Oberndorfer (1921), Schultz (1922) and Schmincke (1922) of giant appendices, and by Pick (1923) of circumscribed overgrowth of a segment of intestine, associated with neurofibromatosis of their nerve supplies.

Moore (1942) pointed out that it is generally accepted that neurofibromatosis is a congenital condition, and that it passes through various phases of development. The increase in endoneural fibrous tissue found by him in cases of macrodactyly in children may be an early stage in the development of neurofibromatosis at a later age. The difference in the rate of development of neurofibromatosis (Ford 1952) would account for the fact that some cases of local gigantism are congenital, whereas others, such as that described here, appear later. The irregular distribution and usual lack of symmetry in bilateral cases are explained by the variable distribution of neurofibromata.

Different views have been put forward to explain the association between neurofibromatosis and local gigantism. Brooks and Lehman (1924) considered that the overgrowth results from invasion of the bone by the neurofibroma, explaining the cases of bone shortening by destruction of epiphysial cartilage. But Moore (1941) stated: " We find it difficult to accept this theory that the bone grows throughout its length, since it runs so directly counter to all recent work on the growth of bone. Furthermore, we can find no report of actual invasion of the shaft by the neurofibroma . . . We believe that overgrowth takes place at the epiphysial line as in normal bone, but at a greatly accelerated rate." It should, however, be noted that Kaplan (1947) described a patient aged twenty-four whose right thumb had become larger during the past two years. Moore (1941) considered that the bony abnormalities result from the changes in the nerves. He stated: " Certainly in these cases there is a defective pattern of growth associated with a defective nerve." He believed that the mixed over-development and under-development that he found were the outcome of faulty control over growth. He found evidence of endarteritis and stated: " Possibly the autonomic nervous system which controls the arteries may also have some control over growth."

Inglis (1950) considered that the elongation of a limb associated with osteochondromata (recorded in two cases by himself and in one case by Bell and Inglis (1925)) and with haemangiomata (described by Bell and Inglis 1925) in an area of neurofibromatous involvement result from neural intrinsic factor of the neurofibromatosis. On this supposition, presumably not only angiomata but also multiple lipomata, which are recorded in association with neurofibromatosis, would have a similar origin.

SUMMARY

1. A case of local gigantism is described, with enlargement of the left thumb, the left ring finger, the right little finger, and the right second and third toes.
2. No other record can be found in the literature of an association of local gigantism of fingers of both hands with a similar condition of the toes of one foot, though there is no theoretical reason why macrodactyly should not affect the digits in any combination.
3. Theories regarding the etiology are discussed.

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REFERENCES

ADAIR, F. E., PACK, G. T., and FARRIOR, J. H. (1932): Lipomas. American Journal of Cancer, **16,** 1,104.
BARALDI, A., and RUIZ, F. R. (1934): Megaloquiria distrófica parcial secundaria a neurinoma del mediano. Revista Médica del Rosario, **24,** 1,243, quoted by Moore, B. H. (1942).
BELL, G., and INGLIS, K. (1925): Plexiform Neuroma. Medical Journal of Australia, **ii,** 423.
BROOKS, B., and LEHMAN, R. P. (1924): The Bone Changes in Recklinghausen's Neurofibromatosis. Surgery, Gynecology and Obstetrics, **38,** 587.
CAPEL, R. H. (1935): Macrodactyly, with Report of a Case. Journal of Anatomy, **69,** 528.
ELKELES, A. (1951): Local Ganglion of Right Third and Fourth Fingers Associated with Multiple Hemangiomata of Right Chest Wall. Proceedings of the Royal Society of Medicine (Clinical Section), **44,** 917.
FORD, F. R. (1952): Diseases of the Nervous System in Infancy, Childhood and Adolescence. Third edition, p. 931. Oxford: Blackwell Scientific Publications.
GUENTHER, quoted by Zondek, H. (1944): The Diseases of the Endocrine Glands. Fourth edition. London: Edward Arnold & Co.
INGLIS, K. (1950): Local Gigantism (A Manifestation of Neurofibromatosis): its Relation to General Gigantism and to Acromegaly. American Journal of Pathology, **26,** 1,059.
KANAVEL, A. B. (1932): Congenital Malformations of the Hands. Archives of Surgery, **25,** 282.
KAPLAN, E. B. (1947): Congenital Giant Thumb. Bulletin of the Hospital for Joint Diseases, **8,** 38.
KILLINGER, quoted by Kanavel, A. B. (1932).
MOORE, B. H. (1941): Some Orthopaedic Relationships of Neurofibromatosis. Journal of Bone and Joint Surgery, **23,** 109.

MOORE, B. H. (1942): Macrodactyly and Associated Peripheral Nerve changes. Journal of Bone and Joint Surgery, **24,** 617.

MOURQUAND and BUCHE, quoted by Kanavel, A. B. (1932).

OBERNDORFER (1921): Partieller, primärer Riesenwuchs des Wurmfortsatzes, kombiniert mit Ganglioneuromatose. Zeitschrift für die gesamte Neurologie und Psychiatrie, **72,** 105, quoted by Winestine, F. (1924).

PICK, L. (1923): Über Neurofibromatose und partiellen Riesenwuchs, insbesondere über die sektorenförmige Kombination von wahrem partiellen Riesenwuchs des Darmes mit mesenterialer Neurofibromatose. Beiträge zur pathologischen Anatomie und zur allgemeinen Pathologie, **71,** 560, quoted by Winestine, F. (1924).

SCHMINCKE (1922): Demonstrationen zur Geschwulstpathologie. Zentralblatt für Allgemeine Pathologie, **33,** 17, quoted by Winestine, F. (1924).

SCHULTZ, A. (1922): Ganglioneuromatose des Wurmfortsatzes. Zentralblatt für Allgemeine Pathologie, **33,** 172, quoted by Winestine, F. (1924).

WEBER, F. P. (1918): Haemangiectatic Hypertrophy of Limbs—Congenital Phlebarteriectasis and So-called Congenital Varicose Veins. British Journal of Children's Diseases, **15,** 13.

WEBER, F. P. (1951): In Discussion of Case Presented by Elkeles. Proceedings of the Royal Society of Medicine (Clinical Section), **44,** 920.

WINESTINE, F. (1924): The Relation of Von Recklinghausen's Disease (Multiple Neurofibromatosis) to Giant Growth and Blastomatosis. Journal of Cancer Research, **8,** 409.

WRIGHT, A. D. (1951): In Discussion of Case Presented by Elkeles. Proceedings of the Royal Society of Medicine (Clinical Section), **44,** 920.

THE REACTIONS OF INJURED HUMAN ARTICULAR CARTILAGE

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The physical and chemical structure of normal hyaline articular cartilage was described by Collins (1949), but there is little published on its reactions to injury and repair in the human subject, though animal observations have been made, notably by Bennett, Bauer and Maddock (1932). The slow metabolism and physiological inactivity of joint cartilage were emphasised by Bywaters (1937), again mainly on animal material, and the general view is that if cartilage has any reactions at all they are so slow that they can be neglected. For two reasons these reactions seem to call for investigation: firstly that in the prolonged course of human arthritis even a slow reaction cannot be neglected; and secondly because the study of the reactions of a substance of relatively known structure under physiological conditions may throw some light on the methods by which the changes of this substance in the arthritic diseases are brought about.

In this paper the changes that follow simple mechanical injuries to the joint cartilage are considered. The affected surfaces were removed at operation or necropsy after intervals from three days to ten years after the original injury; the ages of the subjects lay between twelve and fifty-nine years. The material available included sixteen fractures of the head of the radius, eleven of the patella and smaller numbers of other fractures.

NORMAL STRUCTURE

The terms used by Collins (*loc. cit.*) for the zones of cartilage are self-explanatory. For the wavy line that marks the junction between calcified and uncalcified cartilage—a feature to which reference will frequently have to be made—the term "tidemark," introduced by Fawns and Landells (1953), is conveniently brief.

It is important to keep in the foreground the fact that the articular end of a bone is not a separately made hinge-piece attached to the rest, but is built up in the adolescent epiphysis by growth and ossification over a period of twenty years before it reaches a mature state. In the young child there is no continuous subchondral osseous plate under the thick cartilage, but loci of ossification which come to lie closer together until, in adolescence, a continuous lamella is found; this thickens in adult life in accordance with the stresses and pressures upon it. Only then does a definite zone of calcified cartilage appear beyond the bone. In this zone blood vessels are unusual, though they were described by Holmdahl and Ingelmark in rabbits (1950), and by Harrison, Schajowicz and Trueta in man (1953); they are too few to have much responsibility for cartilage nutrition and are separated from the cartilage by a thin bone lamella. These represent the last traces of active ossification that has been going on since childhood; this process, though dormant, is not effete and can be seen to become reactivated.

Along with the rearrangement of cells and the appearance of subchondral bone that takes place with increasing age, there are conspicuous changes in the cartilage matrix. In the foetus and young child there is little collagen but much polysaccharide; this is in the phase characterised by strong metachromasia with Toluidine blue but a very weak or absent Periodic acid–Schiff reaction. As age advances, the PA–S reaction becomes stronger, the metachromasia less intense and complete, though never lost in health. The collagen component becomes increasingly obvious, staining deeply with van Gieson's stain; but where the metachromasia is deepest and most intense there is less collagen stainable with van Gieson's stain.

It is probably legitimate to suggest that in growth the cartilage cells first form an excessive amount of highly polymerised and sulphated acid mucopolysaccharide. The first fine fibrils of collagen added to this—well below the limit of visibility and hard to demonstrate with the electron microscope—will have their relatively few basic groups saturated with the excess of acid mucopolysaccharide, and so will not show the usual red staining with eosin or with van Gieson; but if the proportion of chondrin to collagen is greatly decreased, whether because too much collagen or too little chondrin is being made, the collagen will then become acidophil. It seems further that at some stage part of the chondrin loses its metachromasia and part—possibly, but not necessarily, the same part—becomes PA–S positive. These changes are commonly seen in older cartilage, both with and without osteoarthritic changes.

The considerable variations in the shape of the cartilage cell, and the extent and shape of the metachromatic patch around it in apparently healthy cartilage, are evidence of the variable physiological state of this cell which is by no means inert when time is granted to its slow processes.

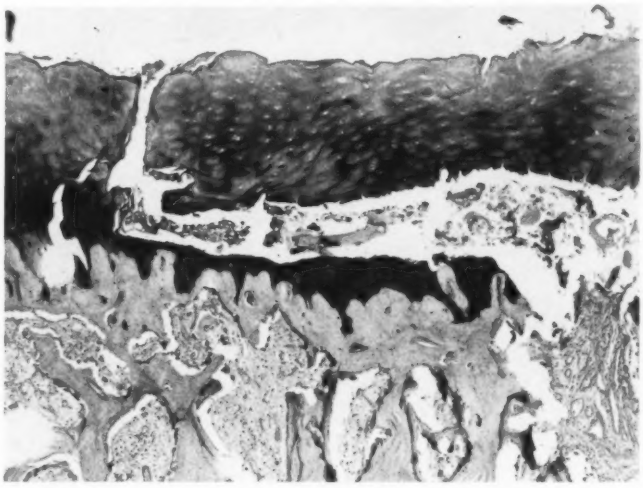

FIG. 1

Pott's fracture, three weeks old. Woman aged twenty-eight. Tibial articular surface. (Periodic acid–Schiff, ×28.) Separation of uncalcified cartilage along the "tidemark"; fibrinous debris in the cleft; bone and calcified cartilage below with callus in the marrow spaces. Fracture completely through bone to the right. Tear in section to the left made during preparation; this remains empty.

OBSERVATIONS ON COMPLETE FRACTURES

Immediate changes:

1. The lines along which the cartilage breaks are those of the fibres described by Benninghoff (1925). Histological features are so often dismissed as artefacts that this independent confirmation of their reality is valuable.

2. In addition to these lines of weakness there is an important additional one lying along the plane of the tidemark (Fig. 1) which is commonly found and has not been commented on. This is the level at which almost all transverse fractures are found. Elsewhere they tend to run vertically through cartilage, calcified cartilage and bone. In particular, there is no tendency to split along the more obvious line of the bone-cartilage junction, where weakness might be expected. This peculiar weakness of the tidemark in one direction is accompanied by increased strength at right angles to it: thus splits from above and erosions from below have a natural

tendency to stop at the tidemark, and if frozen sections are handled this zone may hang together like a cord when the rest of the section is torn. It must be emphasised again that this is not a structurally separately built membrane, but develops in cartilage during adult life as calcification occurs deep to it. The striking difference in physical qualities associated with the adjacent calcification suggests that considerable reorientation of fibres and bonds occurs with calcification.

3. The physical characteristics of plasticity and coherence in uncalcified cartilage are shown well in some fractures. In the specimen shown in Figure 2 the folded piece of cartilage has been torn off at the tidemark, rotated and bent double, so that the original articular surface has been turned towards the interior of the bone. In spite of this violent displacement there is almost no fragmentation.

4. The zone of dead cells around the site of injury is exceedingly narrow; virtually only

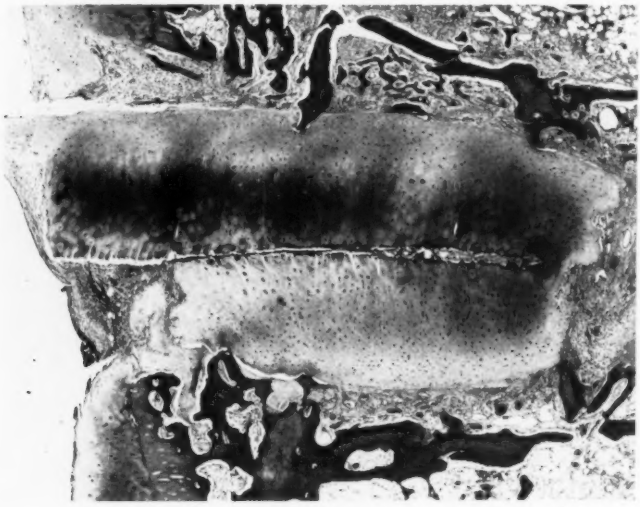

FIG. 2

Fractured head of radjus nineteen days old. Woman aged fifty-six. (Haematoxylin and eosin, ×16.) Extreme displacement, buckling and rotation of cartilage without fragmentation. Early invasion and digestion by granulation tissue below on the left and at the right-hand edge. The dark line of the tidemark can just be made out in the middle.

the cells directly in the line are killed. The elasticity of cartilage is such that brief violent compression is not necessarily fatal.

Intermediate changes—The following observations were made from sections examined three to fourteen days after the injury.

1. There is no tendency for the cartilage to flake around the margin of the injury. This is important in relation to the pathogenesis of osteoarthritis, which is sometimes attributed to disruption of the surface layer, allowing the lower layers to separate. Observation shows, however, that mere mechanical severance of surface layers in healthy cartilage is not of itself sufficient to make the whole incoherent. It will be different if the cartilage is already unhealthy. Longer-term observations where the joint has been subjected to the strain of normal use confirm this: thus, in a shoulder examined seven years after a Nicola's operation for recurrent dislocation, there was no roughening of the cartilage round the opening made in it for the long head of the biceps.

2. There is very little multiplication of the cartilage cells exposed by the breach. Since it is

generally conceded that their nutrition is mainly synovial, it might be expected that the improvement in nutrition brought about by direct access to the synovial fluid would stimulate their multiplication, but even in young humans little of this occurs, though I have seen it to a limited degree in young animals. Here again the import is related to osteoarthritis: the multiplication of cells observed in fibrillated cartilage cannot be explained simply by their improved access to synovial fluid.

Late stage of repair—The following changes were observed in specimens examined fourteen days to ten years after the injury.

Two events require consideration: 1) the replacement of gaps left in the articular cartilage; and 2) the fate of displaced fragments of articular cartilage.

1. Replacement is invariably by fibrous tissue, derived from granulation tissue emerging from the depths of the bone if the gap is central, from the synovial membrane if the gap is

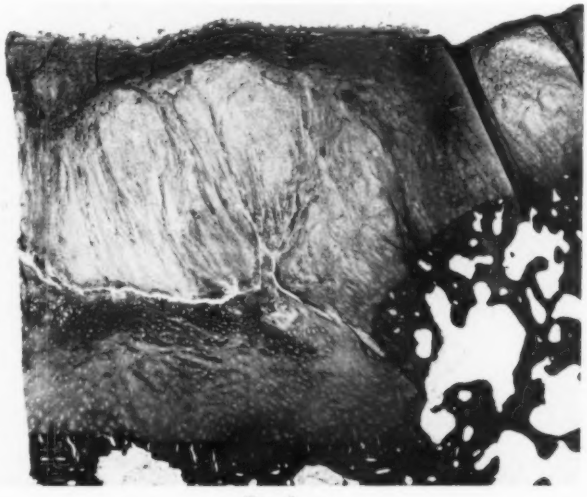

FIG. 3

Pott's fracture seven years old. Woman aged forty-two. Tibial articular surface near medial malleolus. (van Gieson, ×16.) Original articular surface below and to the left, buried by fibrocartilage. Bone to the right formed in repair deep to this.

peripheral, or both. This fibrous tissue, if exposed to the synovial fluid, is eventually impregnated with polysaccharide and may in years come to resemble fibrocartilage and present a remarkably smooth surface, but the internal structure is not that of hyaline cartilage formed since infancy. Repair by genuine hyaline cartilage has not been seen in the present material.

The original hyaline cartilage at the edge of the breach contributes nothing to the new tissue; the line of demarcation is as clear ten years later as it is at the time of the injury (Fig. 3).

When for any reason the synovial fluid cannot reach the fibrous tissue, the resemblance of the fibrous tissue to cartilage is less close—for example the modified fibrous tissue that fills up the space between the cup and the neck of the femur in the operation of cup arthroplasty.

The fibrous cartilage formed in repair is not inefficient, though it has not the structure of the original hyaline. The interlacing coarse-fibre bundles render it less liable to the splitting which disrupts the fine parallel fibres of the usual cartilage in osteoarthritis. Fusion between the new and the old may be intimate, with the immigration of fibrocytes into cartilage matrix, or they may merely lie closely together, with a tendency to split on handling, as in Figure 3.

Bone or calcified cartilage exposed in the joint without fibrous tissue overlying it does not appear to survive. The bone dies when nourished only by synovial fluid, and both bone and cartilage become mechanically eroded and abrade the opposing face. They also form a barrier that granulation tissue is slow to remove, osteoclastic resorption being necessary, and this is seen also in purulent digestion of cartilage. The clinical sequel of osteoarthritis after joint fractures is more attributable to this, to malalignment of the bones, or to changes in the hyaline cartilage of the rest of the joint, than to the fibrocartilage of repair.

2. Displaced cartilage is treated in several ways. When fibrous tissue grows over the surface and is itself nourished by synovial fluid, the new stratum of fibrocartilage may bury the old, which survives almost unchanged beneath it, as seen in Figure 3 seven years after the injury. This is strong evidence of the low energy requirements of hyaline cartilage, here satisfied by diffusion from the joint space through double the usual thickness of material. When, however, the cartilage is displaced into the granulation tissue of the callus, it does not survive. In the first three weeks little appears to be taking place round it, compared with

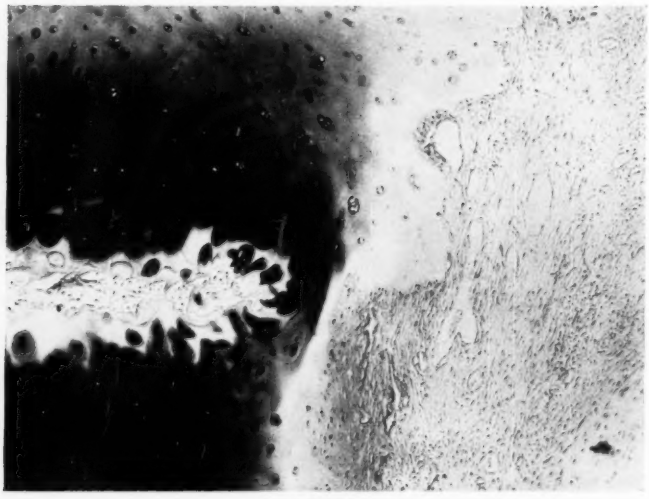

FIG. 4

As Figure 2. Enlargement of the right-hand edge to ×55 to show digestion by granulation tissue from the right.

the activity around fragments of bone; by six weeks the cells are dying, the chondrin leaches away and the fibrous tissue stains increasingly feebly; the cartilage still remains coherent and hyaline, and may be found in that state many years later, with bone deposited over it.

More usually, however, cartilage perishes wherever it is in contact with granulation tissue. The cells die and the chondrin is dissolved away before actual invasion has gone far. The chondrin may be found absorbed on to neighbouring collagen strands; these are metachromatic only when they are close to cartilage that has recently lost its chondrin. Later, immigrant fibrocytes penetrate the matrix and replace it by fibrous tissue, which may ossify. These cells are capable of invading living cartilage, in which case the cells of the cartilage lose their identity but do not always die. More often the cartilage is dead already when it is invaded, the cells undergoing lysis though their spaces may remain long visible. This death appears related to the near approach of the granulation tissue, as the zone of death is distributed around the edge of the cartilage and is greatest near the granulation tissue—exactly the reverse of what would be expected if anoxia or starvation were concerned

(Figs. 2 and 4). This is in keeping with the known low oxygen requirements of cartilage. No foreign cells are required to effect this removal. In calcified cartilage, erosion is associated with the presence of cells like osteoclasts, and no solution of the chondrin takes place (Fig. 5).

It is not possible to associate the death with any particular component of granulation tissue. Polymorphonuclear leucocytes are not concerned in it. The fate of small fragments of cartilage which are dislodged into the joint was described by Lloyd-Roberts (1953); they are swept by movement into the angles of the joint and engulfed by synovial cells.

The evidence from these " experimental human autografts " is the more valuable because serological complications and operative sepsis can be excluded. The violence of the injury remains uncontrolled and, since this will be greatest on the surface of the fragments, it is possible that the death of the cartilage cells may in part precede the arrival of the granulation

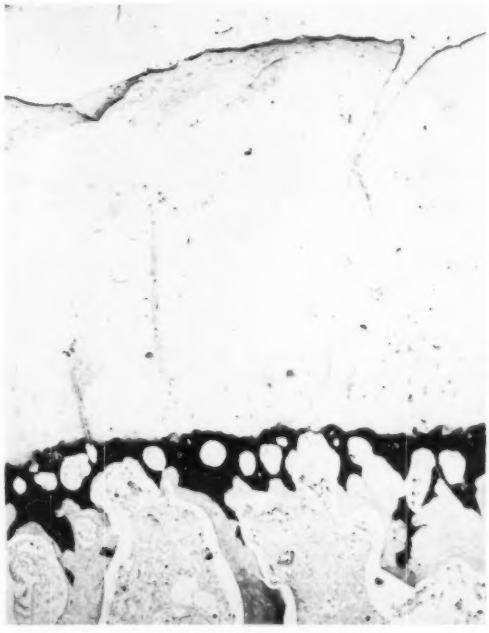

FIG. 5

Fracture of head of femur two months old. Girl aged sixteen. (Toluidine blue, ×38.) Necrosis of cartilage and and bone. Complete removal of chondrin from uncalcified cartilage; no change in the calcified zone.

tissue and may indeed induce the latter. The fact that dead fragments are not seen before the granulation tissue is not incontrovertible evidence that granulation tissue is fatal to cartilage, since chemical changes may precede the visible ones. But the narrowness of the zone of death seen immediately after the injury, and similar changes seen when gross trauma can be excluded (*vide infra*) are stronger evidence.

Bone is often deposited directly on the surface of the displaced cartilage, either alive or dead, calcified or uncalcified. Calcified cartilage, lying deeper, is the more common substrate, but it is of theoretical importance that calcification is not essential. Fine bone slivers can often be seen passing beyond the tidemark. It is often stated that provisional calcification is essential as a preliminary to ossification, and calcification is thought to be indicative of impending ossification. These statements are not always true, and the processes should be

considered as independent though often associated. Calcification is essential for the piecemeal removal of lumps of cartilage by osteoclasts, as distinct from the infiltrating type of destruction by granulation tissue described above. Part of the trouble in the rachitic epiphysis arises from this difference in the removal of the two types of cartilage.

When bone is formed over dead uncalcified cartilage, a haze of calcium granules may be observed to develop in the cartilage. Since there are no visible living cells in the cartilage, this cannot be an active step of deliberate provisional calcification by cartilage cells but must represent a diffusion of calcium from the forming bone. It does not occur in cartilage that has no bone on the surface, and thus cannot be due to absorption of calcium from the blood. It has been suggested elsewhere (Fawns and Landells 1953) that this is the method of formation of the normal calcified zone that lies between the adult articular cartilage and the bony articular lamella.

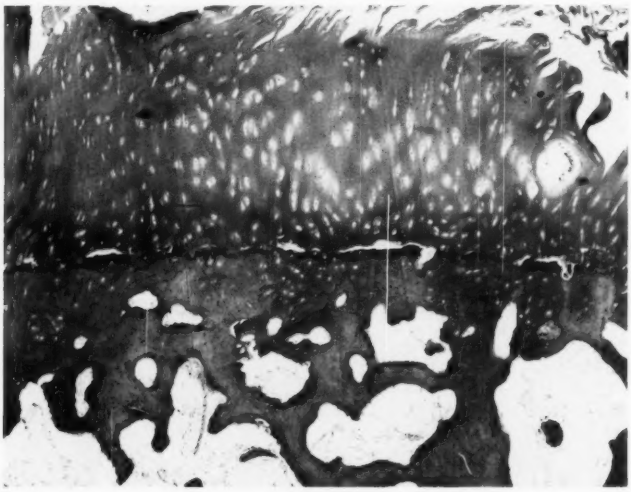

FIG. 6

Fracture of osteoarthritic patella a few days old. Woman aged fifty-three. (van Gieson, ×28.) The fracture is just out of the picture to the right. A cleft containing fibrin-like material crosses nearly the whole section at the tidemark.

OBSERVATIONS FROM INCOMPLETE FRACTURES

When the stress on the joint is insufficient to cause clinical fracture, damage may be found on microscopy, necessarily in areas chosen at random or for other investigations; proof that they are indeed traumatic will be inferential, and it will not be possible to refer the injury to a definite date. Articular cartilage, consisting of an elastic surface layer over a thin calcified brittle one, would be likely to slip at the plane between these layers, and the existence of a plane of weakness at this level has been commented on. Moreover, if there is a gap in the deeper tissue the elastic cartilage might be expected to herniate into the opening. Such changes can be seen in sections, without any other structural abnormality, sometimes near complete fractures but also independent of them, and it is reasonable to suggest that they are mechanical. Three stages, short of complete fracture, can be described: 1) splitting at the tidemark; 2) depression of cartilage into the bone; and 3) fissuring of both calcified cartilage and bone along a vertical plane which permits blood vessels from the marrow to reach the cartilage.

Tidemark splitting (Fig. 6)—These fissures occurring during life are marked by the accumulation of a pool of acellular material; accidental fissures occurring in the preparation of the section are empty. This material has some of the standing features of fibrin: it is strongly PA–S positive, PTAH positive, picrophil in van Gieson's stain; it is not metachromatic, does not dissolve in collagenase, or lose its staining reactions after hyalase.

Impression of cartilage into a vertical fissure (Figs. 7 to 9)—There is usually associated deformity in the adjacent bone and cartilage. The displaced cartilage is unusually deeply stained with basic dyes, unusually resistant to enzyme digestion, and often mixed with the eosinophil material described in the previous paragraph. The cells may either proliferate or atrophy but rarely remain unchanged.

Vascularisation through fissures—Though this is believed to be traumatic in many cases, once the process is established it will closely resemble the picture resulting from reactivation of ossification where it had stopped after adolescence in the articular lamella. In this process the bone that has been laid down will first be breached by osteoclastic resorption, but thereafter traumatic and erosive breaches will look alike, and for that reason the neutral term " intrusion " of blood vessels has been adopted. The consequences of the two events leading to intrusion are similar.

An early but clear example is shown in Figures 10 to 12. The points of particular importance are: 1) Capillaries grow direct into the uncalcified cartilage without the mediation of any other cell to destroy the matrix ahead of them; 2) there is physical splitting of the calcified cartilage behind them, but no invasion or solution of it; 3) the shape of the intruding mass into the cartilage is smoothly rounded; 4) there is well-marked zonation around this—a ring free of chondrin next to the untouched cartilage; next a ring where there is a gathering together of fine fibres to form a coarser set of strands—these are picrophil and strongly metachromatic; and finally among the capillaries the fibres become very large and irregular and the cartilage cells disappear. This zonation is not conspicuous in the PA–S stain. This process and the events taking place around displaced cartilage are similar.

The smooth rounding of the unit suggests that the capillaries act as a whole, not as individual foci of erosion, and also that diffusion is one factor in the process. The simple hypothesis is that there are two fractions of the carbohydrate, a metachromatic mobile one and a fixed PA–S positive one; and that the first result of vascular intrusion is the elution of the chondrin faster than the cells make it, giving the pale ring and permitting the collagen fibres to aggregate into the coarser strands visible in the section. On these coarser strands some at least of the chondrin becomes temporarily readsorbed, so giving the metachromatic-picrophil zone, and preventing the acid dyes from access to the collagen. The PA–S component remains uniformly visible throughout. Under these vascular conditions the cartilage cells either lose their identity or die, the conditions both as regards diffusion currents and oxygenation differing greatly from those in the depths of avascular hyaline cartilage to which they are accustomed.

A rare variation of this picture is shown in Figure 13. There is a dark basophil, but not metachromatic, ring between the pale ring and the zone of twisted " spun " metachromatic strands. This again is related to the focus as a whole and not to any particular component of it; it has the uniformity and smooth outline of a soluble rather than of a particulate material. The intensity of the stain suggests a strongly acidic substance, possibly a modification of the chondrin diffusing one way and meeting other substances coming up from the blood. It is not calcium, which has a granular texture. Like the other changes, it stops abruptly at the edge of the calcified cartilage.

When the process has gone on further, the degeneration of the hyaline cartilage is more complete. In Figure 14 an arteriole is present at the breach through the calcified cartilage. The hyaline cartilage has lost its chondrin and reverted to fibrous tissue, which has broken up into a tuft in the middle of the patellar cartilage very like the fibrillation of osteoarthritis.

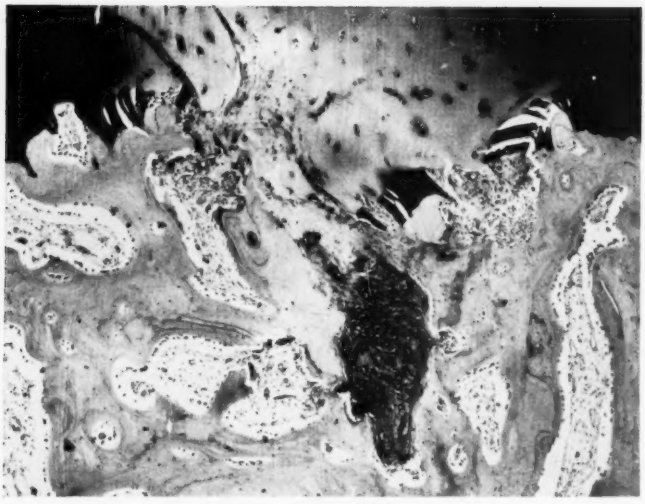

FIG. 7

Fractured head of radius six weeks old. Man aged twenty-seven. (Toluidine blue, ×29.) Gross displacement near line of fracture. Disruption of calcified cartilage and displacement of both calcified and uncalcified cartilage into the bone, the latter proliferating (the tongue of medium grey tissue in the centre).

FIG. 8

Fracture of patella seventeen days old. Man aged twenty-eight. (Periodic acid–Schiff, ×120.) About 1 centimetre from the fracture line; patella apparently healthy. Two gaps in the (slightly paler) calcified cartilage, in both of which uncalcified cartilage is present. Proliferation of cells in the left displacement and ossification in both. Traumatic origin of displacements is likely.

The chondrocytes, as far as they are recognisable in the margins of the intrusion, have reverted to fibrocytes, and there is much more than normal collagen in the area; this process represents not only removal of one component of cartilage but includes excess production of the other.

The usual sequel is the early sealing off of the intrusion by ossification. This results in the formation of a nodule of bone in the deeper cartilage which, by fusion of adjacent foci, advances the bony articular lamella and thins the overlying cartilage in the middle of the joint; this will impair its elasticity and favour further injury. At the edge of the cartilage it results in osteophyte formation.

DISCUSSION

Relation between articular cartilage and blood supply—It was pointed out first by Strangeways (1920) and Fisher (1922), and is indeed a commonplace observation in joint pathology, that cartilage may live, grow and calcify (but not ossify) when completely detached from the bone,

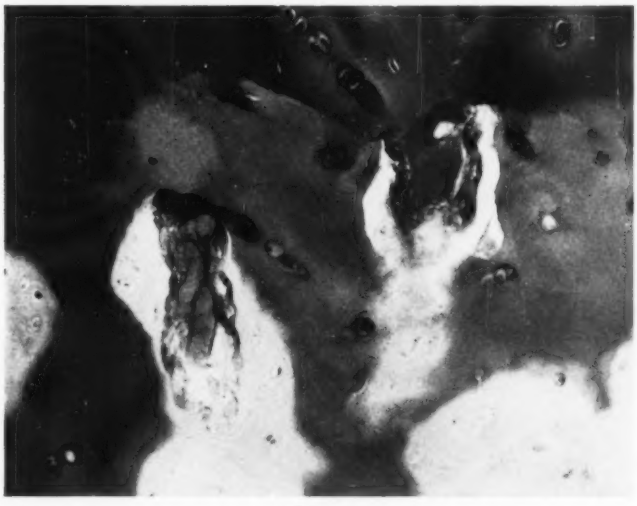

FIG. 9

Patella: apparently normal post-mortem specimen from a man of fifty-three. There was a single small marginal osteophyte and on microscopy a little chondromalacia. No known injury. (Periodic acid–Schiff, ×190.) Gap in calcified cartilage containing debris and uncalcified cartilage. Line of cells of uncalcified cartilage (which should be vertical) is oblique and passes into this area. Dark streak marking abnormal material in tidemark. Presumptive evidence of mechanical displacement.

provided it has free access to the synovial fluid. Because of this and the absence of blood vessels in adult articular cartilage, it is generally accepted that the normal nutrition of cartilage is mainly synovial, though there is evidence of some diffusion from the bone. The presence of occasional blood vessels in the subarticular plane has been interpreted as of importance in the normal nutrition of cartilage, but they are few in number and are insulated by a thin sheet of bone.

If the blood vessels are present without this sheet, abnormalities are visible in the cartilage. Examples of such contact have been given arising in each of three ways—trauma displacing fragments into callus, vascular tissue growing over the surface, and intrusion of blood vessels through the articular lamella from trauma or erosion. In each case the cartilage is being destroyed. It would appear that so far from being necessary for the life of the cartilage, a good blood supply is rapidly followed by complete disorganisation of hyaline cartilage,

and its replacement by fibrous tissue, fibrocartilage or bone. The exiguous nutrition by synovial fluid to which cartilage is adapted is apparently a condition of its health, and the richer standard of living provided by the blood stream is too generous for it. Some confirmation of this view was given by Subba Rao (1954) who observed that cartilage grafts from the xiphisternum of rats were absorbed by granulation tissue within twelve weeks, if they were small. Larger grafts developed a dense perichondrium and persisted; one became ossified.

Mechanism of cartilage removal—No specialised phagocyte appears in the removal of uncalcified cartilage, although when collagen is being removed, for example in suture material or tendon, giant cells are usual. It is possible that the collagen is in fact not removed, but that the solution of the surrounding chondrin changes the physical state of the collagen, so that fine fibrils dispersed in a gel come to form tight bundles (Figs. 10 to 13). These take up little room compared with their immediately adjacent pale ring, possibly explained by the

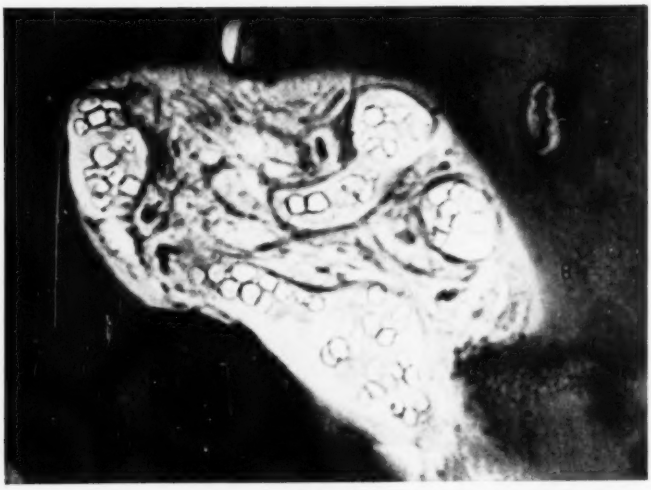

FIG. 10

Patella excised for chondromalacia. Woman aged thirty. (Periodic acid–Schiff, ×530.) The capillaries are close up against the face of the cartilage undergoing solution; five cartilage cells present amongst them but no adventitious phagocytic cells. Expansion of the blood vessels into the uncalcified cartilage above, but limited below by the tidemark.

loss of much bound water as well as the acquisition of fresh interfibrillary bonds. In this vascular digestion the adjacent calcified cartilage undergoes no change; its removal is always associated with the presence of multinucleate giant cells. It is therefore likely that calcium stabilises the linkage between collagen and chondrin in the natural removal of cartilage just as it does in enzyme digestion in experimental conditions (Fawns and Landells 1953).

The relation between collagen staining red with van Gieson's stain and with eosin on the one hand, and collagen without metachromasia and therefore probably free of chondrin on the other hand, suggests that the chemical basis of the attachment of the acid dyes and the acid mucopolysaccharides is the same, and is available for one or other purpose alternatively, but not for both simultaneously. This alternation is seen in arthritic cartilage as well as in the events discussed in this paper. The form and distribution of the PA–S staining material and its digestibility are those of the collagen, and it is therefore suggested that this material, probably polysaccharide, is intimately linked to the collagen.

Relation to osteoarthritis—The frequency with which the changes described as " incomplete fractures " are found is much higher in material that shows naked-eye evidence of osteoarthritis than in joints with a perfectly smooth surface. Trivial defects are common

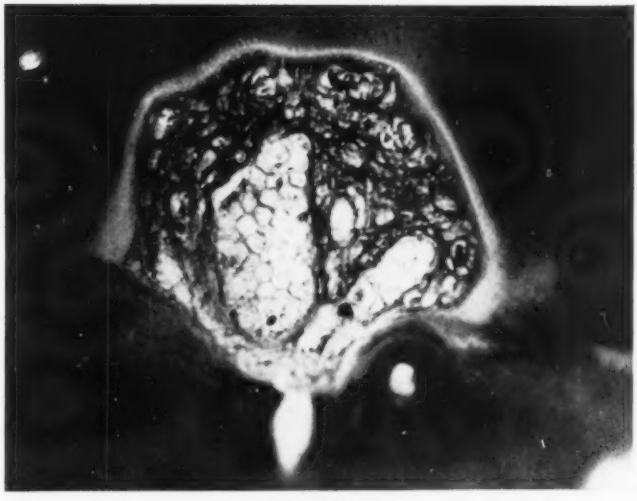

FIG. 11
Another intrusion from the same bone. (Toluidine blue, ×450.)

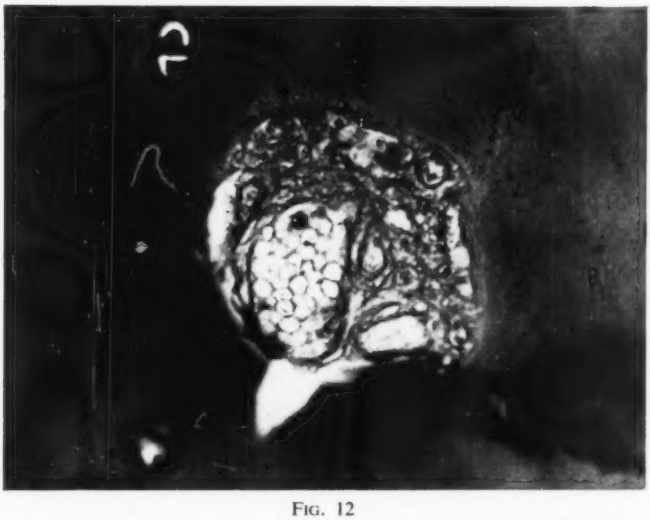

FIG. 12
Serial section. (Periodic acid–Schiff.) The pale ring in the Toluidine blue is obscure in the PA–S. The intrusion is smoothly rounded in uncalcified cartilage but limited by the tidemark, and has broken but not eroded the calcified zone.

enough in controls; severe ones are nearly always found in osteoarthritis, but are not confined to the badly affected parts of the joint surfaces. They can be more easily interpreted as mechanical processes than as either degenerative or reparative. They are clearly associated

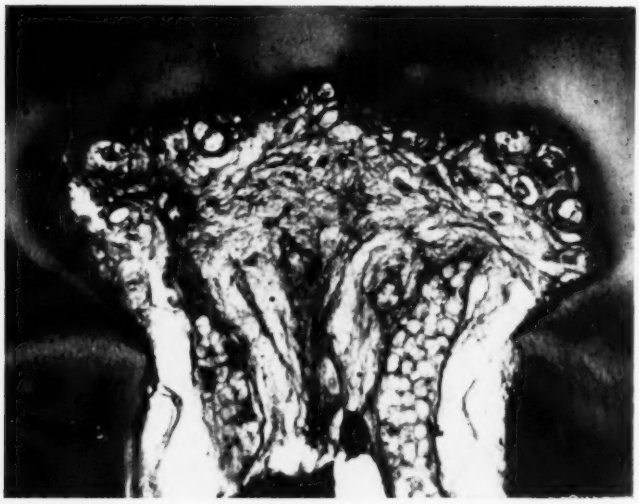

FIG. 13

Another intrusion from the same bone. (Toluidine blue, ×440.) Invasion of cartilage by capillaries from the bone below, with pale diffusion zone limited by a dark basophil ring. Cartilage cells and the aggregation of collagen fibres well seen in the next zone; no adventitious cells. Process mechanically checked in the calcified zone but expands in the soft uncalcified zone; chemical changes stop abruptly at the tidemark.

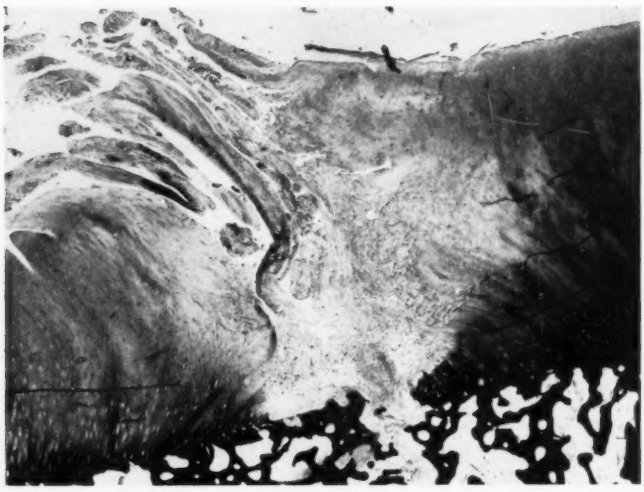

FIG. 14

Patella from a man dead of disseminated lupus erythematosus, aged thirty-four; apparent chondromalacia; no known injury. (van Gieson, ×12.) Gap in the centre through which an arteriole passes. Elution of chondrin; disorganisation of hyaline cartilage and reversion to fibrous tissue in a V-shaped zone centred on the breach. Lines of cartilage-bone junctions on both sides are level; pattern of bone symmetrical; gross fracture is unlikely to have been the cause. Joint elsewhere normal.

with osteoarthritis, but whether as cause or effect is less certain. Minor traumatic events might well have a more serious effect on cartilage that was already degenerate, and inelastic or eroded cartilage will expose the underlying bony lamella to unusual strains which will permit vascular intrusions. The two processes are mingled, but there is some evidence that trauma alone will account for degenerative changes in healthy cartilage, and that frequently repeated minor injuries in ordinary life can so affect the vascularity of cartilage that the changes of osteoarthritis will follow.

Repair of the damaged cartilage of osteoarthritis would require, first, removal of the old material, and second, the free access of vascular tissue to the lacuna. In the body this is denied first by the dense bony lamella which has undergone thickening during the degenerative phase; and if this is penetrated (which is relatively uncommon) the granulation tissue forming will be crushed out of existence by the pressure from the opposite joint surface. Indeed it never achieves more than a local nodule of fibrocartilage and does not flow over the whole bare area. A conspicuous exception to this is the thick fibrocartilaginous cap which forms under the protection of the Vitallium cup in the Smith-Petersen arthroplasty. Here the preparation of the upper end of the femoral neck opens the vascular part of the bone widely and spreads any pressure uniformly. The result is a thick cap of coarse-fibred bluish white material which makes a very effective replacement of the hyaline cartilage, especially when after a few years it is saturated with mucopolysaccharide. The surgical intervention brings to completion the naturally unsuccessful efforts at cure. In the elderly, the vigour of growth is reduced and the available time for the slow repair of cartilage is short; even in young subjects repair is much better seen in the parts of the joint nearest the synovium. This fibrocartilage of repair can be distinguished both on its internal structure and on the appearances in the underlying bone from the degenerative fibrillary changes in osteoarthritis, though it is sometimes confused with it.

SUMMARY

1. The lines of fracture confirm the suggestions of earlier authors on the lines of strength in cartilage, with the additional feature of a transverse plane of weakness at the apex of the calcified zone.
2. The normal nutrition of cartilage is synovial, and access of a free blood supply is followed by destruction of hyaline articular cartilage.
3. Minor traumatic events in the articular lamella are common, particularly in osteoarthritic joints; the results of these on the cartilage are like the changes of osteoarthritis.
4. The removal of uncalcified cartilage can be described in two stages of a physico-chemical kind; the removal of calcified cartilage is a single cellular process.
5. There is evidence that the carbohydrate moiety of cartilage is present in two separable phases, one fixed to collagen, the other free.
6. The repair mechanisms after fracture are those available to restore the damage of osteoarthritis, and reasons can be shown why in fact they are ineffective.

I wish to acknowledge the kindness of the orthopaedic surgeons of the London Hospital for permission to study their material; the similar assistance of Mr N. E. James and Mr M. B. Devas; and the technical and photographic assistance of the staff of the Bernhard Baron Institute.

REFERENCES

BENNINGHOFF, A. (1925): Form und Bau der Gelenkknorpel in ihren Beziehungen zur Funktion. Zweiter Teil: Der Aufbau des Gelenkknorpels in seinen Beziehungen zur Funktion. Zeitschrift für Zellforschung und mikroskopische Anatomie, **2**, 783.

BENNETT, G. A., BAUER, W., and MADDOCK, S. J. (1932): A Study of the Repair of Articular Cartilage. American Journal of Pathology, **8**, 499.

BYWATERS, E. G. L. (1937): The Metabolism of Joint Tissues. Journal of Pathology and Bacteriology, **44**, 247.

COLLINS, D. H. (1949): The Pathology of Articular and Spinal Diseases, pp. 19–23, 59–72, and *passim*. London: Edward Arnold & Co.

FAWNS, H. T., and LANDELLS, J. W. (1953): Histochemical Studies of Rheumatic Conditions. I. Observations on the Fine Structures of the Matrix of Normal Bone and Cartilage. Annals of the Rheumatic Diseases, **12,** 105.

FISHER, A. G. T. (1922): A Contribution to the Pathology and Aetiology of Osteo-arthritis. British Journal of Surgery, **10,** 52.

HARRISON, M. H. M., SCHAJOWICZ, F., and TRUETA, J. (1953): Osteoarthritis of the Hip; a Study of the Nature and Evolution of the Disease. Journal of Bone and Joint Surgery, **35–B,** 598.

HOLMDAHL, D. E., and INGELMARK, B. E. (1950): The Contact Between the Articular Cartilage and the Medullary Cavities of the Bone. Acta Orthopaedica Scandinavica, **20,** 156.

LLOYD-ROBERTS, G. C. (1953): The Role of Capsular Changes in Osteoarthritis of the Hip Joint. Journal of Bone and Joint Surgery, **35–B,** 627.

STRANGEWAYS, T. S. P. (1920): Observations on the Nutrition of Articular Cartilage. British Medical Journal, **i,** 661.

SUBBA RAO, K. V. (1954): An Experimental Study of Regeneration in Cartilage. Journal of Pathology and Bacteriology, **67,** 455.

FEMORAL GROWTH AFTER OCCLUSION OF THE PRINCIPAL NUTRIENT CANAL IN DAY-OLD RABBITS

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Study of the blood supply of bone has as one of its objectives a determination of the relative value of three possible sources of arterial blood to long bones, namely: the periosteal network; the principal nutrient artery; and the small arteries entering the ends of the bones. Since the work of Ollier (1867) on the importance of the periosteum in bone regeneration periosteal arteries entering cortical bone were described (Langer 1876, Testut 1880, Lexer 1922 and Kolodny 1923). Doubt has been thrown on this proposition by Moore and Corbett (1914) who said that the periosteal circulation was of no value in the healing of fractures. In his famous paper (1927) Johnson said that periosteal bone was well and surely laid down only upon a foundation of old bone. He also asserted that there was no bleeding from bone defects after the nutrient and metaphysial blood supplies had been cut off. Drinker, Drinker and Lund (1922), after injection experiments, considered the periosteal circulation of no importance for their subsequent perfusion studies. Brunschwig (1930) was unable to produce any infarction of the tibia after stripping the entire shaft of its periosteum in fifteen adult dogs; and Wu and Miltner (1937) recorded bone lengthening after a similar procedure in young rabbits. Marneffe (1951) stated that only the outer third of rabbit bone cortex is dependent on a periosteal blood supply. Recently the very existence of periosteal arteries in rabbit cortical bone has been denied by Brookes and Harrison (1957) who, from their radiological studies, envisaged a copious venous drainage route in the microscopic vascular channels passing centrifugally from the cortex out into the periosteal capillary bed.

It is accepted here that a periosteal arterial supply in the rabbit is a negligible factor in the vascularisation of long bones. In view of the importance attributed by anatomists and surgeons to the role of the principal nutrient artery in bone nutrition it is wise to make a functional comparison of this artery against the small arteries that enter the extremities of the long bones (Hunter 1743). An attempt has been made in this paper to gain this by obliterating the principal nutrient canal of the femur in day-old rabbits so that it is dependent solely upon its epiphysial and metaphysial arteries for a supply of blood, and observing the subsequent growth of the bone.

MATERIALS AND METHODS

Day-old rabbits were anaesthetised with open ether, and an incision was made on the medial side of the thigh from the inguinal groove to just above the medial femoral condyle. With blunt dissection the femoral bundle and lateral circumflex femoral artery were revealed. Arising from the root of this, the principal nutrient artery was readily followed in the interval between the vastus medialis and adductor group of muscles distally to its entry into the principal nutrient canal of the femur, situated just below the lesser trochanter.

Although it is a simple matter to ligate the nutrient artery alone, leaving the vein intact, it was felt that permanent occlusion of the canal was the procedure of choice because of the possibility of regeneration of the ligated nutrient artery into a patent canal. The consequent disturbance of the venous side of the circulation in the femur would be negligible in view of the profuse venous drainage at the bone extremities (Langer 1876, Harrison and Gossman 1955) and at the surface of the diaphysis (Brookes and Harrison 1957).

The technique of occlusion was based on the work of Nakahara and Dilger (1909) on heterotopic bone formation. The periosteum and diaphysial surface in the immediate neighbourhood of the canal were curetted and the scrapings packed into it, the nutrient bundle being destroyed in the process. The operation field was closed by three blanket sutures, the incision painted with proflavine solution, and the rabbit returned to the nest after it had recovered from the anaesthetic. Subsequent bone production by the displaced periosteal fragments brought about complete bony occlusion of the canal, as judged by inspection of the cleaned adult femora at necropsy.

The rabbits were in four separate litters. Occlusion of the nutrient canal was carried out on right or left femur or both femora, each litter retaining its own controls. After the initial mortality among the young, the four litters yielded three, six, six and eight animals respectively, the growth of whose femora could be followed for five months until they reached maturity.

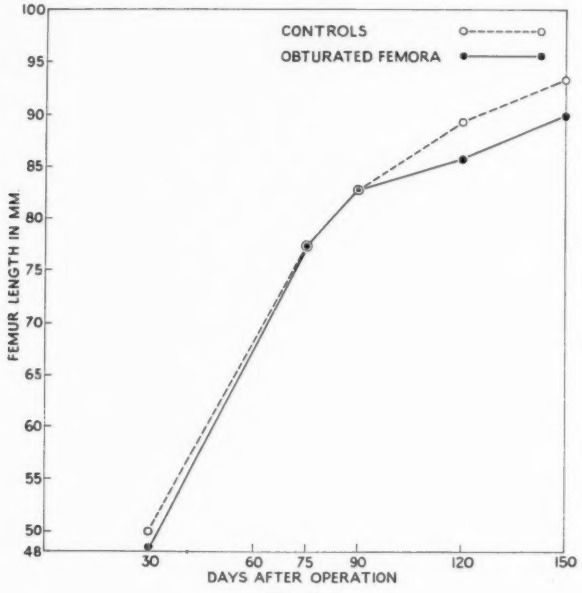

FIG. 1
Growth curves of rabbit femora.

Femur length was measured indirectly at 30, 75, 90, 120 and 150 days after the operation. From radiographs of the hind limbs applied as closely as possible to the film, the greatest distance was measured with calipers between the femoral head and the mid-point of a line joining the distal surfaces of the condyles. A radiographic method for measuring bone growth in goats was used by Bisgard (1936) whose results imply an accuracy sufficient to detect differences of 0·5 millimetre.

One hundred and fifty days after operation the animals were killed by intravenous nembutal, and the arteries of the hind limbs were injected with a barium sulphate suspension (" Micropaque," Damancy and Co.) through the abdominal aorta. After fixation in 5 per cent formol-saline the femora were cleaned of all extra-osseous tissue and their greatest length measured directly with calipers.

The cleaned femora were placed in a 5 per cent solution of nitric acid in 5 per cent formol-saline. The decalcified specimens so obtained were radiographed on Kodaline film so that the intra-osseous arteries of the bones operated upon and control bones could be visualised.

RESULTS

The direct and indirect measurements of femur length at stated intervals during the growing period are set out in Table I. The effect of occlusion of the nutrient canal on femoral growth was calculated for each period from the data in the columns by means of the analysis

TABLE I

FEMUR LENGTH IN MILLIMETRES, RABBIT DATA

Litter	Rabbit	Side operated upon	Length in millimetres (indirect measurements)										Length in millimetres (direct measurements)	
			30 days		75 days		90 days		120 days		150 days		150 days	
			Right	Left	Right	Left	Right	Left	Right	Left	Right	Left	Right	Left
1	1	(Control)	43·5	43·3										
	2	Left	43	40·8	67	66	75·5	73	82	79	80	76	86	85
	3	(Control)	48	48	75	74	78·5	76·8	83	81·3	88	86	97·5	86
2	4	Right	53·8	51·3	81	81·5	77·3	78	84·5	86	88·3	90·5	88·5	90
	5	(Control)	53	52	79	77·5	83·5	82	90·5	89	92	92	91·5	91
	6	Right	50·4	50·5	78	79	82·5	83·5	87·5	87	94	95·5	91	92
	7	R. and L.	49	47·5	71	71	76·5	75	84·5	80·5	86·5	84	85·5	84·2
	8	(Control)	55·3	55										
	9	R. and L.	51	50	74·3	73	73	75·5	84·3	83	88·5	87·8	88·5	87·5
3	10	(Control)	54·5	53	80	79·5	85	83	94	94·5	98	98	97·5	97·5
	11	Right	50	51	75	76	83·3	84·5	89·5	89·8	94·3	95	93·5	95
	12	(Control)	50		78	76·5	83·5	83·3	93	93·5	96	96	96	96
	13	Right	49	52	80	81·3	83·5	84·3	91·5	91·5				
	14	Right	46	47	77·5	77·5	84·5	85·3	92	95·3	96·5	101	98	100
	15	Left	51·3	51	81	80	86·3	85	98	96·5	102	100	99	98
4	16	R. and L.	48·5	47·8	75	79·5			83	85·5	83	85	86	91
	17	Right	44·5	50	80·3	80·3	86	87						
	18	R. and L.	44·5	46	76·5	79	82·5	84·5						
	19	Right	46·5	46·5	79·5	80			86	85	88	87·5	90	90·5
	20	(Control)	50	49	79	75·5	83·5	81						
	21	Left	46·5	44·3	78	78·3			82	83	88·5	88	92	90·5
	22	(Control)	52·5	53·5	81·5	83			90	89	93	93	95	95
	23	Left	51	50	76·5	76	83	82·5						

of variance, including the operation of occlusion and leg-side as sources of variation. Figure 1 shows that the femur of a newborn rabbit deprived of its principal nutrient artery has an almost normal growth curve. A significant deficiency in length of the order of 3 per cent only

occurs during the initial and final phases of growth, while in the intermediate phase of seventy-five to ninety days there is no significant difference in length between occluded and control femora.

To test the validity of the indirect measurements from radiographs, the direct and indirect data for right and left femora at the close of the experiment at 150 days were compared by means of Student's " t " test. The comparison yielded t=0·3 and 0·43 (P=0·8 and 0·7) for the right and left sides respectively, showing that the radiographic data have the same value in a large group of observations as direct measurements of the cleaned bones. The mean

FIG. 2

FIG. 3

Figure 2—Arteriograph of the distal part of a rabbit femur 150 days after occlusion of the principal nutrient canal, showing intra-osseous metaphysial anastomoses. (×3·5.) Figure 3—Arteriograph of the distal part of a normal rabbit femur. (×3·5.)

deficiency in length as calculated from direct measurements at 150 days is 77 per cent of that obtained from an analysis of indirect data at the same period (Table II). Because indirect measurements seem to magnify the length differences when present, all mean differences calculated from indirect data have been reduced in this ratio before the percentage diminution in length at the various time intervals has been determined and the growth curves for control and occluded femora have been constructed.

Radiological examination of the injected bones shows that the medullary arteries of occluded specimens are well filled and that the general vascular pattern is but little disturbed.

Filling has taken place through epiphysial and metaphysial arteries which have undergone enlargement. Above the condyles conspicuous intra-osseous anastomoses are seen which are not normally present (Figs. 2 and 3), and the artery of the trochanteric fossa at the proximal end of the femur has greatly enlarged and become an important supply channel to the medullary arterial system (Figs. 4 and 5).

DISCUSSION

Occlusion of the nutrient canal eliminates the functional activity of the nutrient vein and sympathetic nerve fibres from the growing femur besides that of the principal nutrient artery. The effects of removal of the nutrient vein may be discounted for the reasons already given, and interruption of an automatic pathway to the bone may be taken as of no effect because all experimental investigations of lumbar sympathectomy and ganglionectomy on bone growth in animals have had negative results (Bisgard 1931, 1933, Wilson and Thompson 1939).

TABLE II

DIMINUTION IN LENGTH OF THE ADULT RABBIT FEMUR AFTER OCCLUSION OF THE PRINCIPAL NUTRIENT CANAL IN THE NEWBORN

Days after operation	Diminution in length (*millimetres*)	Corrected diminution (*millimetres*)	Mean femur length (controls) (*millimetres*)	Percentage diminution
30	2·1	1·6	50·0	3·1
75	0	0	77·2	nil
90	0	0	82·6	nil
120	4·6	3·5	89·2	3·9
150	4·4	3·4	92·8	3·7
150	3·4 (Direct measurement)	3·4 (Direct measurement)	93·2 (Direct measurement)	3·7

A fundamental factor in osteogenesis is an adequate blood supply. Variations in nutrition of the growth cartilage will cause shortening or lengthening of long bones—for example the hypertrophy of limb bones associated with arterio-venous aneurysms (Horton 1932, Chandler 1937). Ferguson (1933) first stated that interruption of the medullary arteries and diversion of blood to the growth cartilages accounted for the increased growth rate observable in long bones affected by a variety of pathological conditions, such as fractures, chronic infections and tumours. Occlusion of the nutrient canal may result in diversion of blood into the epiphysial and metaphysial arteries, with possible ischaemia of the diaphysis. Kistler (1934), who blocked many arterioles with large carbon particles, produced some fibrosis of the central marrow. Huggins and Wiege (1939), who avulsed the principal nutrient artery and vein, found only a temporary anaemia of bone marrow, which after seventy days was histologically normal. It is probable that, while a collateral circulation is developing in the bone extremities, there is a diminution in the femoral blood supply and to the growth cartilages, thus accounting for the growth lag of the occluded femora in the first thirty days.

With the establishment of a collateral circulation by means of the anastomoses between the metaphysial arteries and the principal nutrient artery (Figs. 2 and 4) the blood flow near the growth cartilages is increased, bringing about equalisation in length of occluded and normal femora in the intermediate growth phase. In the final phase, 120 to 150 days, there appears to be a relative decrease in the nutrition of the growth cartilages to account for the 3·7 per cent retardation in femoral growth at maturity. The reason for this is not clear. It seems probable that towards the end of growth the collateral circulation is not quite able

to furnish the same quantity of blood to the medullary arterial system as when a nutrient artery is also available as a supply channel. Nevertheless, the collateral circulation in metaphysial intra-osseous anastomoses is 96 per cent functionally adequate; consequently the dictum of Harris (1929) that diaphysial terminal arteries are end-arteries cannot be accepted.

The medullary arterial system, fed by nutrient, metaphysial and epiphysial arteries, is to be regarded as the vascular mechanism for the delivery of blood to long bones. In the rabbit

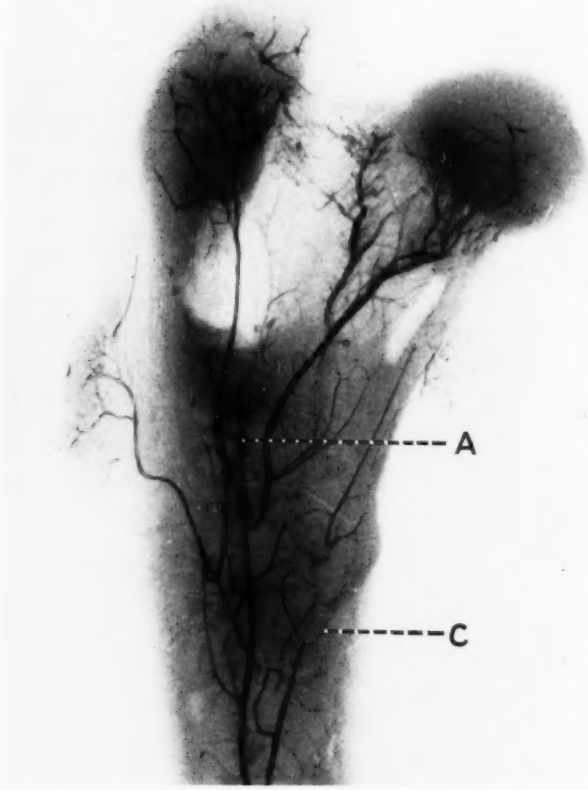

FIG. 4

Arteriograph of the upper part of a mature rabbit femur. The principal nutrient canal, whose site is indicated at C, was occluded 150 days previously, twenty-four hours after birth. The artery of the trochanteric fossa, A, has taken over the blood supply of the upper end of the femur and become a main supply channel to the medullary arterial system. (×4.)

the principal nutrient artery is not of vital importance because the medullary arterial system can rapidly accommodate itself to dependence on the metaphysial arteries alone.

These results may be compared with the evidence for bone lengthening after fractures in children, in whom presumably the same local vascular mechanism is active that determined the growth curve of the occluded femora. Many authors have reported this occurrence (Truesdell 1921, Burdick and Siris 1923, David 1924, Chandler 1937). Although decrease in

shortening, or occasional actual lengthening of an affected long bone has been described, the patients have been kept under observation for a comparatively short time, usually less than two years after fracture, and offered data generally fall within the ages four to fourteen years. The clinical evidence supports the finding of stimulation of osteogenesis in the intermediate growth phase. Since epiphysial fusion in the tibia and femur occurs at from eighteen to twenty years, it seems unwise to assume, in view of the terminal retardation in growth found

FIG. 5
Arteriograph of the upper end of a normal femur of an adult rabbit. The principal nutrient artery, A, divides into ascending and descending branches on entering the medulla. (×4.)

experimentally, that shortening due to overriding fractures of the femur and tibia in children will be annulled at maturity. There is no clinical survey to show this.

Femora may behave differently from tibiae after a diaphysial disturbance to the medullary arterial system. Bisgard (1936) found that overriding fractures caused an initial relative diminution, subsequent lengthening and final shortening of the tibia in one goat, but a final lengthening in its twin. Trueta (1953), working on rabbit tibiae, was convinced that by prolonged

interruption of the nutrient and periosteal circulation of the shaft he was able to increase bone growth. It is conceivable that exclusion of the tibial principal nutrient artery might cause a more prolonged increase in vascularity of the growth cartilages, because there is no artery supplying the tibia comparable with the artery of the trochanteric fossa, a large vessel which is at some distance from the superior growth cartilage of the femur.

It was first pointed out by Grégoire and Carrière (1921) that the human femur has two principal nutrients. This might allow for discrepancies in behaviour of children's femora and tibiae after fracture.

SUMMARY

1. The principal nutrient canal of the femur in day-old rabbits was occluded and subsequent femoral growth observed.
2. An initial shortening is followed by equalisation and a final absolute shortening of the order of 3 per cent in occluded femora as compared with controls.
3. It is emphasised that the medullary arterial system, fed by principal nutrient, metaphysial and epiphysial arteries, is the mechanism for the delivery of arterial blood to long bones, and that the arteries of bone extremities are of overwhelming importance in the nutrition of the long bone as a whole.

It is a pleasure to acknowledge the help and advice I have received from Professor R. G. Harrison in the preparation of this paper. My thanks are due to Mr L. G. Cooper and Mr A. Taunton for their technical assistance with the photography.

REFERENCES

Bisgard, J. D. (1931): Effect of Sympathetic Ganglionectomy Upon Bone Growth. Proceedings of the Society for Experimental Biology and Medicine, **29,** 229.
Bisgard, J. D. (1933): Longitudinal Bone Growth. The Influence of Sympathetic Deinnervation. Annals of Surgery, **97,** 374.
Bisgard, J. D. (1936): Longitudinal Overgrowth of Long Bones with Special Reference to Fractures. Surgery, Gynecology and Obstetrics, **62,** 823.
Brookes, M., and Harrison, R. G. (1957): The Vascularization of the Rabbit Femur and Tibiofibula. Journal of Anatomy, **91,** 61.
Brunschwig, A. (1930): Experimental Infarction of Bone Marrow. Proceedings of the Society for Experimental Biology and Medicine, **27,** 1,049.
Burdick, C. G., and Siris, I. E. (1923): Fractures of the Femur in Children. Annals of Surgery, **77,** 736.
Chandler, F. A. (1937): Local Overgrowth. Journal of the American Medical Association, **109,** 1,411.
David, V. C. (1924): Shortening and Compensatory Overgrowth Following Fractures of the Femur in Children. Archives of Surgery, **9,** 438.
Drinker, C. K., Drinker, K. R., and Lund, C. C. (1922): Circulation in the Mammalian Bone Marrow. American Journal of Physiology, **62,** 1.
Ferguson, A. B. (1933): Surgical Stimulation of Bone Growth by a New Procedure. Journal of the American Medical Association, **100,** 26.
Grégoire, R., and Carrière, C. (1921): Circulation arterielle intraosseuse du fémur et du tibia. Comptes Rendus de l'Association des Anatomistes à Toulouse, **16,** 179.
Harris, H. A. (1929): The Vascular Supply of Bone, with Special Reference to the Epiphysial Cartilage. Journal of Anatomy, **64,** 3.
Harrison, R. G., and Gossman, H. H. (1955): The Fate of Radiopaque Media Injected into the Cancellous Bone of the Extremities. Journal of Bone and Joint Surgery, **37-B,** 150.
Horton, B. T. (1932): Hemihypertrophy of Extremities Associated with Congenital Arteriovenous Fistula. Journal of the American Medical Association, **98,** 373.
Huggins, C., and Wiege, E. (1939): The Effect on the Bone Marrow of Disruption of the Nutrient Artery and Vein. Annals of Surgery, **110,** 940.
Hunter, W. (1743): Of the Structure and Diseases of Articulating Cartilages. Philosophical Transactions, London, **42,** 514.
Johnson, R. W., Jun. (1927): A Physiological Study of the Blood Supply of the Diaphysis. Journal of Bone and Joint Surgery, **9,** 153.

KISTLER, G. H. (1934): Sequences of Experimental Infarction of the Femur in Rabbits. Archives of Surgery, **29,** 589.

KOLODNY, A. (1923): The Periosteal Blood Supply and Healing of Fractures. Journal of Bone and Joint Surgery, **5,** 698.

LANGER, K. (1876): Über das Gefässsystem der Röhrenknochen, mit Beiträgen zur Kenntniss des Baues und der Entwicklung des Knochengewebes. Separatabdruck aus dem XXXVI Bande der Denkschriften der Mathematisch-Naturwissenschaftlichen Classe der Kaiserlichen Akademie der Wissenschaften. Wien: Aus der Kaiserlich-Königlichen Hof- und Staatsdruckerei.

LEXER, E. (1922): Über die Entstehung von Pseudarthrosen nach Frakturen und nach Knochentransplantationen. Archiv für klinische Chirurgie, **119,** 520.

MARNEFFE, R. DE (1951): Recherches morphologiques et expérimentales sur la vascularisation osseuse. Brussels: Acta Medica Belgica.

MOORE, J. E., and CORBETT, J. F. (1914): Studies on the Function of the Periosteum. Surgery, Gynecology and Obstetrics, **19,** 5.

NAKAHARA, T., and DILGER, A. (1909): Subkutane und intramuskuläre Knochenneubildungen durch Injektion bezw. Implantation von Periostemulsion. Beiträge zur klinischen Chirurgie, **63,** 235.

OLLIER, L. (1867): Traité expérimental et clinique de la régénération des os et de la production artificielle du tissu osseux. Paris: V. Masson et fils.

TESTUT, L. (1880): Vaisseaux et nerfs des tissus conjonctifs fibreux, séreux et osseux. Paris: Octave Doin. Thèse d'agrégation.

TRUESDELL, E. D. (1921): Inequality of the Lower Extremities Following Fractures of the Shaft of the Femur in Children. Annals of Surgery, **74,** 498.

TRUETA, J. (1953): The Influence of the Blood Supply in Controlling Bone Growth. Bulletin of the Hospital for Joint Diseases, **14,** 147.

WILSON, P. D., and THOMPSON, T. C. (193?): A Clinical Consideration of the Methods for Equalizing Leg Length. Annals of Surgery, **110,** 992.

WU, Y. K., and MILTNER, L. J. (1937): A Procedure for Stimulation of Longitudinal Growth of Bone. An Experimental Study. Journal of Bone and Joint Surgery, **19,** 909.

CHARLES BELL KEETLEY

(1848–1909)

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Charles Robert Bell Keetley was born on September 13, 1848, in the same year as Sir William Macewen and a little before Arbuthnot Lane (1856), Robert Jones (1857) and A. H. Tubby (1862). In a generation of brilliant men and at a time of striking surgical progress Keetley's own place in the development of surgery has been rather overshadowed. Yet he made several original contributions, particularly in orthopaedics, and was an able exponent of the best of the new surgical methods. Treasurer of the British Orthopaedic Society and author of a useful text-book, he was an enterprising surgeon, pioneering in operation for Pott's paraplegia and adolescent coxa vara.

EARLY YEARS

He was born in Grimsby, Lincolnshire, where his father, Robert Keetley, was a shipbuilder and at one time Mayor. During his early education at Browne's School he acted as unarticled apprentice to his uncle, Thomas Bell Keetley, a surgeon practising in Silver Street, Great Grimsby. He later studied botany and anatomy at the Hull school of medicine, entering St Bartholomew's Hospital as a medical student in 1871. He was a keen athlete during his student days, excelling at football, rowing and boxing. He obtained the diplomas of M.R.C.S. and L.R.C.P. in 1873 and the following year was awarded two gold medals in the Intermediate London M.B. He did not go on to complete the final examination for his degree but took a post at the Queen's Hospital, Birmingham, as House Surgeon. In 1875 he entered general practice for a short time at Bungay, Suffolk.

He soon returned to London, being appointed Demonstrator of Anatomy at St Bartholomew's in 1876, the year he obtained his F.R.C.S. During the next two years his work included experience in the orthopaedic department under Alfred Willett and Howard Marsh successively.

THE WEST LONDON HOSPITAL

In 1878, the year after Lister came to London, Keetley was elected Surgeon to the West London Hospital at Hammersmith, with which he remained associated for the rest of his life. He was one of the first to recognise the value of the new antiseptic methods, the practice of which he introduced at this hospital. He carried out its detail with such success that at one time some of the larger London hospitals insisted upon candidates for the appointment of

house surgeon visiting the West London Hospital in order to perfect themselves in the technique. The development of the hospital from a small suburban venture into a great charity to which became attached a flourishing school of medicine was largely owing to Keetley's initiative and energy. In 1882 he was one of the founders and first President of the West London Medico-Chirurgical Society. He also originated, with H. W. Chambers, the Army Medical Civilian Reserve which was afterwards merged into the Territorial Force as the nucleus of the Third London General Hospital during the first world war.

He was a voluminous writer, contributing many valuable papers chiefly on orthopaedic, abdominal and plastic surgery. One of the earliest of his publications was a small book entitled *The Student's Guide to the Medical Profession* which appeared in 1878 and outlined medical training and careers.

His *Index of Surgery* (1881) was a handbook designed for use by the senior student just before his final examination. It comprised an alphabetically arranged surgical text largely derived from Keetley's own notes made for the Fellowship examination. Students then, as now, were grateful for brevity and the book went into four editions in six years. In 1883 he published anonymously a book for the lay public, entitled *Kallos, a Treatise on the Scientific Culture of Personal Beauty and the Cure of Ugliness*. Good looks are to be achieved, the reader gathers, by the adoption of simple rules of hygiene and the very occasional use of local applications.

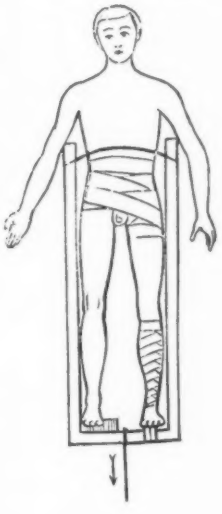
ANNALS OF SURGERY

Keetley was one of the founders of the *Annals of Surgery*. As his co-editor, Lewis S. Pilcher of Brooklyn, wrote later, it was " during an after dinner talk in London in the spring of 1884 that the idea of a surgical journal which would comprehensively represent both British and American surgical work was discussed between us." When the *Annals* appeared the following year Keetley contributed the first article in the first issue. He also wrote the last article in the fiftieth number to round off his service to a journal which, although now entirely an American concern, initially anticipated the present international collaboration that goes to make the *Journal of Bone and Joint Surgery*.

FEMORAL OSTEOTOMY

In 1884, six years after Macewen had introduced supracondylar osteotomy of the femur for genu valgum, Keetley described his experiences with osteotomy of the upper end of the femur, on six occasions supratrochanteric, on four subtrochanteric. The operation was performed for ankylosis of the hip in poor position and was carried out by a " blind " technique. After operation the use of a simple wooden splint was advised. This apparatus is the same in principle as the " well-leg " traction of Roger Anderson, and held the hip well in the desired position of abduction or adduction. In later years he followed Watson Cheyne in using thickly silver-plated steel pins which " are extremely useful in fixing bony fragments after either fracture or osteotomy; but they should never be trusted alone."

In 1887, in an address to the West London Medico-Chirurgical Society, he was able to contrast the safety with which the knee joint



Keetley's apparatus for use after osteotomy of the upper end of the femur. " Little blocks of wood, not unlike an octavo book in size and shape, are placed between the crosspiece of the splint and the sole of the foot on the sound side, and the patient is told to keep the knee of this leg straight. In this way and without any other bandaging, the angle of abduction or adduction of the hip operated on can be regulated so nicely that a little abduction can be made to exactly compensate for an inch or so of real shortening. Extension is not always required." (From the *British Medical Journal* 1884, **i**, 263.)

could be subjected to operation compared with ten years before. He was mainly concerned with the treatment of tuberculosis, for which he advised excision of diseased synovial membrane and curetting of bone. " It is not necessary to remove absolutely every vestige of disease in order to effect a cure." The value of Esmarch's tourniquet and post-operative splinting was stressed.

ADOLESCENT COXA VARA

In 1886 Monks of Boston described what was probably the first recognised example of this condition. He realised that his patient, a boy with bilateral hip disease, had elevation of the trochanters relative to the femoral heads, but he also believed the femoral heads to be dislocated and the whole due to arthritis deformans. Two years later, in September 1888, Keetley published the next account of the disease. He judged the true nature of the trouble rather more accurately, although attributing it to rickets. He was the first ever to treat the deformity by operation.

His patient was a girl aged twenty who had first noticed prominence of the right hip about eight years before. The paper is illustrated by clinical photographs which show well the marked adduction deformity, shortening of the leg and eversion. The patient had already seen a number of surgeons who had offered a variety of diagnoses including sarcoma, periostitis and dislocation. Keetley wrote: " I became convinced that the case was really one of rachitis adolescentium attacking first the upper epiphysial region of the right femur." On March 9, 1888, he removed at operation a wedge of bone from below the greater trochanter, tenotomised the adductor longus, and snapped the femur laterally, greatly diminishing the apparent shortening.

In 1900 he read a paper on coxa vara to the Medical Society of London in which he reviewed the considerable amount of German work that had been done since the condition was first described. He was able to give an excellent clinical description of the disease and an account of the macroscopic changes in the femoral neck. He described, although it does not appear that he had performed, a rather more elaborate subtrochanteric osteotomy of a geometric type, aiming at lengthening the lower margin of the femoral neck as well as correcting the neck-shaft angle.

BRITISH ORTHOPAEDIC SOCIETY

At the Annual Meeting of the British Medical Association in Bristol on August 3, 1894, Keetley, Robert Jones and five others met to discuss the formation of a British Orthopaedic Society. The Society held its inaugural meeting on November 3, 1894, when Keetley was elected Honorary Treasurer. This post he held throughout the Society's existence. He also played a prominent part in the clinical meetings and discussions. Although at one time there were forty members the Society did not survive. The last meeting recorded was held on February 15, 1902.

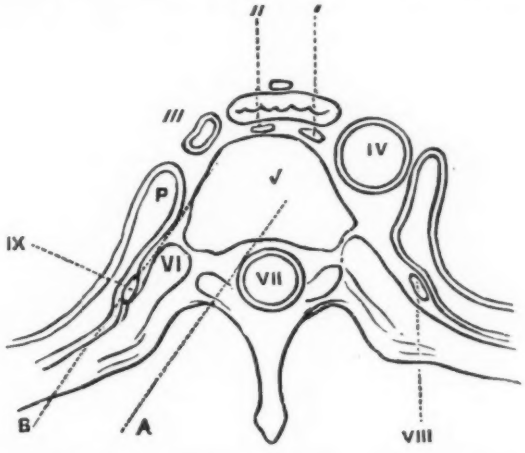
Keetley was a corresponding member of the American Orthopaedic Association.

SURGICAL TREATMENT OF POTT'S DISEASE

Laminectomy in tuberculosis of the thoracic spine came into vogue about 1882, initially with the object only of relieving paraplegia. As Keetley wrote in 1900, " the results did not satisfy many surgeons who were aware of what time and fixation (applied properly) could do for these cases. A large proportion of the cases operated upon died more or less soon after the operation, though not perhaps as a consequence of it, and of those cases in which relief followed, it was obviously doubtful, in a considerable proportion, if the cure were due to the operation."

Keetley went on to describe the operation of laterally trephining the vertebral bodies, anticipating by forty years Capener's antero-lateral decompression of the spinal cord. " The following case is an example of this procedure and was the first, so far as I know, in which it was followed: Frank F——, aet 14, of Topplesfield, Halstead, Essex, had suffered for

four years from paraplegia, practically complete. He came under my care in 'Accident' Ward, West London Hospital, early in 1890, and I applied under extension a Sayre's plaster-of-Paris jacket. In a few days he was able to stand, and even drag himself round the ward with the aid of his hands and the chairs, tables, etc. But no further improvement took place. Therefore on September 19th I removed the laminae of three middle dorsal vertebrae. At the same time I trephined the body of the vertebra corresponding to the angle of the curvature by perforating the transverse process and pedicle (on the right side, in a direction from without inwards and forwards). I used some rather large centre-bits made especially for the purpose and worked by an ordinary drill, enlarging the canal thus bored by means of a gouge and sharp spoon. I did not find any tuberculous focus, or granulations, or pus. In fact I believe the caries, which was of old standing, had ended in cicatrisation and ankylosis. The wound healed by first intention, but even after several months the patient was no better as regards



OUTLINE DIAGRAM BASED ON PART OF A DRAWING IN GRAY'S 'ANATOMY.' Between the straight dotted lines B and A lies the postero-lateral path of access to the bodies of the middle dorsal vertebræ (viewed from below). I, thoracic duct; II, right pneumogastric nerve; III, vena azygos major; IV, aorta; V, vertebral body; VI, rib-head; VII, spinal canal; VIII, left sympathetic nerve; IX. right sympathetic.

The illustration accompanying Keetley's account of his operation for Pott's paraplegia.

his paralysis than on his admission and worse than when merely treated by suspension and Sayre's jacket. He went home into the country on April 23, 1891. Two or three years afterwards I met the Rev. Dr Taylor of Banstead, who agreeably surprised me by saying my operation had cured the young man, who was then walking about well and strongly. I do not think his recovery was due to the operation at all."

Keetley advised the operator to have a dry vertebra or two before him. "The head and neck of the neighbouring rib may be trenched upon without scruple . . . When there has been much destruction and little cicatrisation, granulations, debris and perhaps pus should be quickly discovered. Then a blunt spoon will be safer and more useful than a sharp one." The evacuation of pus and curettage of diseased bone could also be achieved by following and widely opening the track of a sinus or abscess. Keetley was convinced of the value of these procedures and the importance of instilling a bactericidal agent—in his day iodoform—at the site of disease.

ORTHOPAEDIC SURGERY: A HANDBOOK, 1900

Towards the end of the nineteenth century a number of orthopaedic text-books appeared, of which perhaps A. H. Tubby's *Deformities: a Treatise on Orthopaedic Surgery* (first edition 1896) later became the best known. Keetley published in 1900 a text-book of his own, " not as one who supplies a great want but rather as a statement of the views and as an analysis of the observations of a surgeon who has for twenty-two years devoted much time, thought and labour to studying deformities and practising their treatment." He accused the earlier leaders of orthopaedic surgery of being too ready to put forth plausible hypotheses almost exactly as if they were proved theories. " I felt a desire to remove some of the dust in which mistaken ideas of what constituted proof had gradually been hiding simple facts about the pathology and treatment of deformities—of scoliosis for example."

On the last subject he vehemently contradicted the views supported by Tubby and Arbuthnot Lane, among others, that the deformity could be caused by occupational or postural strains. " Once again let us try to realise how utterly unscientific is the practice of saying 'so-and-so used to carry a heavy bag with one hand, or to regularly practise on the violin, and *consequently* he got lateral curvature of the spine.' How surgeons can talk and write like this when any evening they may, if in London, go to a theatre and see a dozen straight-backed musicians, who have fiddled away for their daily bread from boyhood upwards, is really astonishing." Keetley concludes " Before leaving this subject I should like to mention that scoliosis occurs in animals, such as horses, which walk on all fours, and also in fishes. A scoliotic goldfish was to be seen swimming in a bowl for some years in ' Accident ' Ward at the West London."

It illustrates the rapidity with which Röntgen's discovery of x-rays in 1895 was put to use that in this book, published only five years later, there are eight reproductions of radiographs, including a pair demonstrating the fallacies of retouching (" nine skiagrams out of ten reproduced in medical papers and books are faked up in this way ").

Keetley was sceptical of attempts then being made by other London surgeons to correct paralytic talipes by tendon transposition. " The results as far as I have had the opportunity of seeing them, have not been particularly good." Nevertheless, he did himself attempt to correct paralytic calcaneus by transfer of the tibialis anterior and peroneus longus into the tendo calcaneus. In the design of leg irons and calipers it is apparent from the illustrations that distressingly little progress has been made with these in the last fifty years.

As with Tubby's text-book there is no account of disease of the peripheral bones and joints, although both writers mention the close relation these conditions have to the surgery of deformities.

Keetley's enterprising attitude to operative surgery has already been illustrated. The account of surgical treatment in spinal caries was taken from this book. He fully realised, however, the frequency of spontaneous recovery in tuberculosis and the tendency to attribute the cure to whatever treatment was being used at the time. " Time changed the fashions, sent them out, and once in a while brought them in again; and while he thus amused himself, it was this old, but not altogether unkindly enemy who was slyly effecting most of the cures himself."

Rickets, genuine and surmised, was a main preoccupation of the orthopaedic surgeon at the turn of the century. " Drunkenness does not contribute to the accident ward so much as rickets does to the orthopaedic." Although its pathology was not fully understood, on treatment Keetley was able to write: " the remedy about whose value we are most agreed is cod-liver oil."

THE KEETLEY-TOREK OPERATION

On April 16, 1896, at a meeting of the Medical Society of London, Keetley showed two patients to demonstrate a method of retaining the testis in the scrotum in operation for

imperfect descent. The testis was sutured to the inner aspect of the thigh through incisions in the adjacent skin over scrotum and leg. This procedure, re-invented and popularised by Torek in 1931, has since been widely adopted.

Keetley was one of the first to advocate and practise operation in acute appendicitis. In 1900 he described the extra-peritoneal method of draining an appendix abscess. He later introduced the practice of appendicostomy to this country. In fact his belief in the occasional value of conserving the appendix led him into considerable controversy with William Mayo in the pages of the *Annals of Surgery* during 1909.

A letter from Keetley to the *Lancet* in 1904 called attention for the first time to the risk of injury to the chauffeur in the act of starting a car. " I have seen many cases myself. Usually it is a sprain of the wrist, or a bruise or laceration of the back of the hand caused by a ' back-fire.' The wrist is sometimes actually fractured." Ever progressive, Keetley advised: " it is quite easy for any automobilist to do as I have done and have his car fitted with a starter which will not suffer from backfire and which can be operated even by a lady or a child."

He married Anna, daughter of Henry Holmes Long of the Honourable East India Company, but had no children. He died of cardiac syncope on December 4, 1909, at Brighton and was buried at Kensal Green. Had it not been for his slight but incurable deafness and lack of business habits he might have risen to greater heights in his profession. He was habitually unpunctual and was also perhaps handicapped by a facile pencil and fatal gift for impromptu rhyming which led him to make caricatures sometimes with biting effect. He was, however, according to Plarr (1930), easily first in the second rank of London surgeons, an excellent operator, a great teacher and a fine organiser.

I am very grateful for help and encouragement from Mr J. Crawford Adams. I wish also to thank Dr Maurice Shaw, Dean of the West London Hospital Medical School, for his generous assistance.

REFERENCES

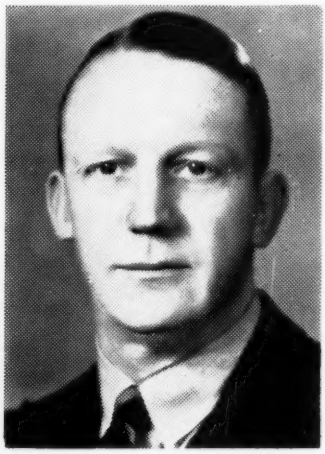
BRITISH MEDICAL JOURNAL (1909): **ii,** 1,721.
DICTIONARY OF NATIONAL BIOGRAPHY SECOND SUPPLEMENT (1912): *Sub nomine.*
KEETLEY, C. B. (1878): The Student's Guide to the Medical Profession. London: Macmillan.
KEETLEY, C. B. (1881): An Index of Surgery. London: Smith, Elder & Co.
KEETLEY, C. B. (1883): Kallos. London: Simpkin, Marshall and Co.
KEETLEY, C. B. (1884): Osteotomy of the Hip. British Medical Journal, **i,** 261.
KEETLEY, C. B. (1888): A Case of Rachitis Adolescentium. Illustrated Medical News, **1,** 7.
KEETLEY, C. B. (1894): Two Cases of Retained Testis Presenting Points of Special Interest. Transactions of the Medical Society of London, **17,** 349.
KEETLEY, C. B. (1900): A Mode of Operating in Cases of Appendicitis. Lancet, **i,** 87.
KEETLEY, C. B. (1900): Orthopaedic Surgery: a Handbook. London: Smith, Elder & Co.
KEETLEY, C. B. (1904): Motor Cars for Medical Men. Lancet, **i,** 551.
LANCET (1909): **ii,** 1,788.
PILCHER, L. S. (1910): Annals of Surgery, **52,** 850.
PLARR'S LIVES OF THE FELLOWS OF THE ROYAL COLLEGE OF SURGEONS OF ENGLAND (1930): *Sub nomine.*
WEST LONDON MEDICAL JOURNAL (1910): **15,** 69.

IN MEMORIAM

SELWYN B. MORRIS
1906-1956

The death of Selwyn Bentham Morris at Auckland on June 20, 1956, after several months of ill health deprived New Zealand of one of its most distinguished and popular orthopaedic surgeons.

Selwyn Morris was educated at King's College, Auckland, and qualified in medicine in 1929 at the University of Otago, Dunedin. As an undergraduate his versatility and wide interests were apparent—he passed his first professional examination with distinction, won the junior clinical medical medal, and the Christie medal for applied anatomy, and was a capable cricket and hockey player, winning his Blue at hockey.



After two years as a resident at Auckland Hospital he went to England to further his surgical studies, and while there worked at the Seamen's Hospital, Greenwich, the West London Hospital, and the Royal Surrey County Hospital, Guildford.

After obtaining his English Fellowship he spent a year at the Royal National Orthopaedic Hospital where he came particularly under the influence of the late Mr A. S. Blundell Bankart, and this he found a valuable period in his training. After this he spent some time in Vienna studying the work of Böhler, and a number of the ideas of that school were brought back here.

On his return to New Zealand in 1936 he took his Australasian Fellowship and was appointed Assistant Orthopaedic Surgeon to the Auckland Hospital, a few years later becoming a Senior Visiting Orthopaedic Surgeon, a position which he held until his death.

Selwyn Morris's energy, his enthusiasm for his work, his knowledge and his ready wit at once endeared him to all those with whom he came in contact. His ability was such that many demands were made on him and he served as a member of the Auckland Hospital Board and of the Auckland University Council, and was co-author of booklets on educational matters and on hospital organisation and reform.

He was unfit for military service during the war, but he had charge of orthopaedic cases in the Military Hospital at Auckland, and during that time he became well known among the American surgeons stationed here, and found much pleasure from meeting them again when he visited the United States in 1951.

He devoted a great deal of time to the Crippled Children's Society, and this type of work he seemed to enjoy most. He was a Fellow of the British Orthopaedic Association and a foundation Fellow of the New Zealand Orthopaedic Association and its first Editorial Secretary; he was also an examiner for the Royal Australasian College of Surgeons.

At medical meetings his papers were always concise and thoroughly prepared, and those on spinal fusion presented to the New Zealand Orthopaedic Association in 1950, and on the management of tuberculosis of the spine in New Zealand at the 1953 meeting of the Association, will be particularly remembered.

Despite all these and other activities, and a busy private practice, Selwyn Morris maintained an active interest in all branches of sport and enjoyed a most happy home life with his wife, four sons and a daughter. His loss will leave a gap for many years. O. R. N.

PROCEEDINGS AND REPORTS OF UNIVERSITIES COLLEGES, COUNCILS AND ASSOCIATIONS

GREAT BRITAIN

BRITISH ORTHOPAEDIC ASSOCIATION

ROBERT JONES CENTENARY MEETING

The Robert Jones Centenary Meeting of the British Orthopaedic Association was held at Shrewsbury and Oswestry on May 9–11, 1957. The President, Mr Philip Wiles, was in the chair.

The results of spinal fusion for scoliosis—*Mr I. W. Winchester* (Oswestry) had reviewed the results in sixty-six cases considered suitable for fusion in the last twenty years at Oswestry, at first under the direction of Mr McCrae Aitken and latterly under Mr Osmond-Clarke. These made up 7 per cent of the patients with scoliosis seen. Fusion was performed to diminish final deformity or to hold correction, to stabilise the spine, and to prevent pain. The Risser jacket had now been abandoned for the Abbott method. After fusion patients spent three to six months recumbent and a further six months in an ambulatory jacket. Eleven patients followed for less than two years were excluded. Of the remaining fifty-five, thirty-three had idiopathic scoliosis, eleven had congenital scoliosis and eleven had paralytic scoliosis. Eighty per cent had relapsed, including all those in whom some previous correction of the curve had been obtained. Quoting correction as a percentage of the original curve, the average original correction was 38 per cent, the final maintained correction 14 per cent. Mr Winchester accounted for this relapse rate under four headings: 1) inaccurate measurements; 2) failure to produce adequate compensatory curves—the danger of over-compensation was probably only theoretical; 3) subsequent growth, with failure to extend the fusion into the compensatory curves; and 4) development of pseudarthroses, although none was detected in this series. He concluded that correction of rotation was a vital factor in preventing relapse, and that the object of plaster correction was to increase the compensatory curves rather than to correct the primary curve.

Normal and abnormal movements of the spine with special reference to scoliosis—*Mr Robert Roaf* (Liverpool) recalled that the usual account of spinal movements was that the spine was a tube of unequal flexibilities in which the movements of lateral flexion and rotation were inevitably linked, that the axis of rotation was the centre of the vertebral body, that rotation did not occur in the lumbar region, that lateral flexion did not occur in the thoracic region and that scoliosis was an exaggeration of a normal movement due to an increase in " unequal flexibilities."

From a study of fresh specimens and radiographic examination of children, it was concluded that, in the child, all these statements were erroneous. The normal young spine was a tube of equal flexibilities, lateral flexion and rotation occurred quite independently, and in both the thoracic and the lumbar region.

The common " rotation-type " of scoliosis was not an exaggeration of a normal movement: it could not be reproduced by artificial limitation of spinal flexibility. It was due to a combination of a torsion and asymmetry of the neural arch and an abnormal relationship between the vertebral bodies. As a result of this, a number of forces—the erector spinae muscles, gravity, the deformed rib cage, and posterior spinal fusion—all tended to increase the deformity. In the rotation type apparent lateral flexion was largely lordosis and efforts at correction should be concentrated on de-rotation.

Mr J. C. Scott (Oxford) stressed the need to be sure of the indications for an operation involving some risk to life and considerable morbidity.

The usual anatomy of posterior dislocation of the shoulder—*Mr W. Gissane* (Birmingham) recalled that Astley Cooper had described posterior dislocation as easily diagnosed, but in fact it was a rare lesion and the diagnosis was often missed. Fractures of the humeral head, either a coronal split or a " pond " fracture, from contact with the posterior rim of the glenoid, were common and produced harsh crepitus. From dissection and operation findings it was clear that the glenoid labrum remained intact, the capsule being torn close to it. The dislocation was due to a medial rotation force; the posterior glenoid margin might also be fractured. The subscapularis remained intact. The lesion had been explored through a posterior incision, splitting the posterior third of the deltoid and dividing infraspinatus. Fractures of the head had been wired—the wire lying under the articular cartilage—

and the dislocation reduced by lateral rotation of the arm. The patient could return to work in three months, but it might be a further year before full movement was recovered. *Sir Reginald Watson-Jones* (London) said that the essential point in reduction was lateral rotation and that the arm must be splinted for three weeks in lateral rotation to prevent recurrence. *Mr Gissane* thought that fixation in lateral rotation was not necessary after open reduction and repair; the shoulder was stable in a sling.

Freiberg's infraction (Köhler's second disease)—*Mr I. S. Smillie* (Dundee) noted that reports on this subject had been sparse since Freiberg's original paper in 1914. Forty-one cases were reviewed, of which three were bilateral. Thirty-one patients were female, ten male. Right and left feet were equally affected. He maintained that they occurred in structurally weak feet with short, varus or hypermobile first metatarsals, and that they therefore had the same basic etiology as march fractures. Both were really stress fractures, and indeed two of his adult patients had associated march fractures. He described the stages by which this stress fracture in the metatarsal head progressed. The central and dorsal part of the head, at first attached by a plantar hinge, finally separated completely and became a loose body depressed in a cavity, the lateral margins of which later gave way and collapsed. This process as seen in radiographs had been confirmed at operation, and he considered the process comparable to that occurring in osteochondritis dissecans in the knee.

The first stage should be treated by immobilisation. In later stages, as long as the plantar hinge remained intact, he recommended that the lesion should be explored, the depressed fragment levered forward and the gap filled with cancellous bone. His results demonstrated almost perfect restoration of the contour of the metatarsal head and recovery of normal bone texture. Attempts to reproduce these results once the fragment had completely separated had not been successful. In all cases an anterior arch support should be used. *Mr H. Jackson Burrows* (London) illustrated the formation of a dorsal osteophyte in early Freiberg's disease and the subsequent changes, and demonstrated the similarity of the lesion in the lunate bone in some cases of Kienböck's disease. He remarked that similar focal lesions were sometimes seen in the femoral head.

Visit of the 1956 Travelling Fellows to the United States and Canada—*Mr D. L. Evans* (London) gave a report of the tour, illustrated with colour transparencies of such brilliance and beauty as to fill his audience with envy.

Congenital vertical talus—*Mr J. Rowland Hughes* (Oswestry) said that although the diagnosis could be suspected at birth, confirmation could only be obtained radiographically. With the foot at right angles to the leg the talus lay in the same line as the tibia and the calcaneum was in equinus and projected in the sole of the foot. The head of the talus lay medially, wedged between the calcaneum and the navicular, which articulated with the dorsum of the neck of the talus. There was contracture of the anterior soft parts, particularly the tibialis anterior, and the spring ligament was elongated. The ankle joint capsule became invaginated into the joint between the talus and the navicular, and added to the soft-part obstruction to reduction. The normal medial deviation of the neck of the talus was exaggerated. Treatment of the late neglected case was so difficult and unsatisfactory as to warrant drastic measures at the earliest stage. In the early case maintaining the foot in full equinus usually corrected the dislocation at the talo-navicular joint, which however commonly recurred when the foot was brought back to the right angle. He had therefore tried scarifying the talo-navicular joint with a tenotome at the time of reduction, and also the use of Steinmann pins to maintain correction after open reductions, both with some success. Lengthening of the tendo calcaneus had potential dangers. He had not tried Osmond-Clarke's method of inserting the peroneus longus into the corrected talus, but had on occasion done an osteotomy of the neck of the talus, maintaining an open wedge on the plantar aspect by a bone graft. He thought that the defect might arise as a further stage of whatever condition in utero produced a talipes calcaneus. It might depend on the tightness or otherwise of the tendo calcaneus. He thought that the partial interposition of the ankle joint capsule into a new talo-navicular joint suggested an acquired lesion, but the presence at birth of a well marked articular facet on the neck of the talus showed that the lesion had been present in utero for some time. *Professor Bryan McFarland* (Liverpool) believed that the lesion was a congenital dislocation. The age at which treatment was started was vital; if left it was a crippling condition, and he had found any manipulative treatment after walking had started most disappointing. *Mr W. Sayle-Creer* (Salford) agreed that it was a true congenital dislocation. It was the soft parts that prevented reduction or caused it to recur, and surgical treatment should concentrate on this factor. *Mr H. Osmond-Clarke* (London) pointed out that the lesion was not very rare. Treatment must start before the age of two; otherwise alteration in the shape of the body of the talus prevented reduction because the talus became impacted in the ankle mortise on attempting dorsiflexion. *Mr Hughes* agreed that early treatment was vital, but patients did not all respond to manipulative methods. He did not believe that it was a primary dysgenesis of the talo-navicular joint, but a secondary lesion as proved by the formation of a secondary articular facet.

Epiphysial stapling in idiopathic knock-knee—*Mr A. E. Pritchard* (Liverpool) had studied twenty-six patients treated by Mr Robert Roaf at Liverpool and by the surgeons of the Robert Jones and Agnes Hunt Orthopaedic Hospital. Conservative treatment had failed to benefit these children, who were all eight years of age or more and almost all overweight. He maintained that surgery was occasionally indicated in this type of patient and believed that epiphysial stapling was superior to supracondylar osteotomy, because 1) the cosmetic result was better; 2) the period in hospital was shorter, and 3) nursing was much easier.

The operative technique was described, and he stressed that no stripping of periosteum was necessary. It was pointed out that it was probably unnecessary to staple the upper tibial epiphysis in addition to the femoral. Three staples must be used and should be left in place until there was slight bow leg; it was not possible to predict the time required for correction in each individual, which varied from five to twenty-one months. The results were excellent in twenty-four patients; relapse followed in two. One patient was left with some loss of knee movement. After removal of staples there was a sudden spurt of growth, followed by normal epiphysial growth. In no instance had this failed to follow. Tribute was paid to Dr Walter Blount who pioneered the procedure. *Sir Reginald Watson-Jones* (London) said that in the last twenty years he had never found it necessary to operate on a patient with knock-knee. Correction always took place within one to three years if Robert Jones's principle was followed; shoes with inner wedges on the crooked heel and sole must be worn constantly—even in the bathroom. *Mr W. Sayle-Creer* (Salford) denied that wedges always cured knock-knee, and thought that the use of a night splint was essential. Stapling operations were liable to leave swollen knees with ugly scars, and he preferred a supracondylar osteotomy when necessary. *Mr Robert Roaf* (Oswestry) said that all the cases discussed by Mr Pritchard had been deteriorating in spite of conservative treatment. There was no difficulty in removing the staples as long as they were not buried under the periosteum. He now rarely felt it necessary to staple the tibia also. *Mr H. Osmond-Clarke* (London) thought that splints were useless and inflicted psychological trauma on the child. His experience led him to use staples less and less in this condition.

Tissue immunity factors in bone homografting—*Mr J. Chalmers* (London) had undertaken experiments to see whether bone homografts participated in a tissue immunity reaction.

Bone homografts to a muscle site in rats gave rise to an early phase of new bone formation which was arrested at ten days; subsequently the new bone died. After several weeks a second phase of new bone growth appeared in relation to the graft.

If the rat was previously immunised by a skin graft from the same donor, the early phase of new bone growth did not occur. Similarly, if the homograft was killed by freeze drying, the early phase did not occur, although the second phase did develop in a few instances. It was concluded that the early phase of new bone formation arose from osteogenic cells carried over with the graft, which became the victim of an immune response by the host. The second phase was due to metaplastic induction in the host connective tissue cells.

Further evidence was provided by experiments in which skin homografts were carried out in animals which had been previously grafted by fresh bone homografts from the same donors. The skin grafts underwent an early rejection at six days. When the bone homograft had previously been treated by freeze drying subsequent skin homografts survived the normal span of eleven days. This showed that bone contained tissue antigens and that both bone and skin had antigens in common. These antigens were inactivated by freeze drying.

It was concluded that bone bank homograft was unlikely to prove inferior clinically to fresh homograft, because neither could make a contribution of living cells. As dead graft did not stimulate immunity, some other process than a tissue immunity reaction must be responsible for the differences in behaviour between bank homograft and fresh autograft. *Dr Paul Curtiss* (Cleveland), while agreeing with the conclusions of Mr Chalmers, reminded the audience that as yet no circulating antibody active against a homograft had been identified, so that the question of immunisation was not yet proven.

Adolescent hallux rigidus—*Mr A. Lipmann Kessel* (London) described an operation that he had studied with *Mr G. L. W. Bonney*. He said that although symptoms would usually retrogress under conservative treatment, some cases were resistant. Extension at the metatarso-phalangeal joint was markedly limited, but the range of flexion remained unaffected. To transpose this range of movement to a useful arc they had performed an extensive osteotomy of the base of the proximal phalanx as close as possible to the articular cartilage, excising a small wedge with its base on the dorsum and leaving the cortex and periosteum on the plantar surface intact. This gap was closed by forcing the toe into dorsiflexion, where it was immobilised for four to six weeks. In all but one of eight patients symptoms had been completely relieved and to the authors' surprise they found that the total range of movement had been increased in each case. One adult patient so treated was also relieved. Their follow-up was so far limited to three years. *Mr R. I. Stirling* (Edinburgh) thought that the essential point was to ensure

that the pressure of weight bearing through the joint was returned to the thicker central articular cartilage rather than borne by the thinner periphery. He advised a plantar capsulotomy with division if necessary of the short flexor tendon and immobilisation in full dorsiflexion for four weeks. *Mr E. Mervyn Evans* (Swansea) stressed that in adolescence hallux rigidus was much commoner in girls than boys, the reverse of the adult sex incidence. In the few adolescent cases that had failed to respond to prolonged conservative therapy he had found hydrocortisone injections most efficacious against pain. *Mr F. C. Dwyer* (Liverpool) thought the operation a good one but doubted whether there was a real increase in the range of movement. *Mr Lipmann Kessel* said he had no explanation to offer for the curious sex incidence. Longer observation was necessary to assess the true value of the operation.

Spontaneous atlanto-axial dislocation, a complication of spondylitis—*Mr D. W. Purser* and *Dr J. Sharp* (Manchester) had studied this problem. Although it was very rarely reported in the literature they had seen forty examples, seventeen in ankylosing spondylitis and twenty-three in rheumatoid arthritis. The condition was liable to produce tetraplegia. The diagnosis depended on flexion-extension radiographs, the lateral view revealing the forward shift of the atlas on flexion, which was associated with rupture of the transverse ligament. In ankylosing spondylitis it occurred usually in long-standing and often still active cases, many with severe spinal flexion deformity and some with signs of cord involvement. The head was tilted forward, with flattening of the angle between neck and occiput; pain was made worse by coughing, and radiotherapy gave no relief. The onset of neurological symptoms might be sudden, and the presence of lesions in the lower cervical spine might complicate the diagnostic problem. Reduction and fixation by bone grafting from occiput to axis usually led to almost complete recovery. In some cases the lesion became stable spontaneously. In rheumatoid arthritis a painful click was often present on movement, but neurological involvement was much less common. The authors considered that the presence of nerve lesions was an absolute indication for operation, and pain if sufficiently severe. Reduction was attained, and maintained during operation, by traction with skull calipers inserted farther forward than usual. Three weeks after operation a plaster was applied embodying the skull calipers and was maintained until union was sound. *Mr J. H. Annan* (Huddersfield) showed radiographs of a similar case with gross displacement when the patient was erect which was reduced spontaneously in the supine position.

Non-union of the epiphysis of the lateral condyle of the humerus—*Mr C. C. Jeffery* (Exeter) stated that ten of thirteen cases of non-union which he had investigated had followed quite minor displacements. The other three had followed open reduction and soft-tissue suture of major displacements. Not one was the result of a major displacement that had remained untreated.

It was shown that some fractures with initially slight displacement progressed to non-union, with increasing deformity leading to grossly distorted elbows. It was also noted that non-union with minor displacement could lead to delayed ulnar palsy.

As a result of the investigation the following recommendations were made. 1) After open reduction the fragments should be stabilised by internal fixation with a stainless steel screw to maintain exact reposition; simple suture was insufficient. 2) Imperfect closed reductions should be critically assessed with a bias towards open reduction and screw fixation. 3) Union must be established unequivocally by radiographs. 4) When union was delayed, or when non-union without significant displacement had developed, a drill hole should be made through the part of the diaphysis that had separated with the displaced epiphysis and on into the humeral diaphysis, the resulting cavity being packed with iliac cancellous bone. This procedure had resulted in bony union, so minimising deformity, in the three cases of non-union which arose while under supervision. *Mr J. N. Wilson* (London) referred to the unexplained problem why non-union occurred. He stressed that the fragment was displaced forwards with the radial head as well as laterally, and that there was some lateral subluxation of the whole forearm. *Professor Bryan McFarland* (Liverpool) thought the fracture was unstable because the fragment carried with it half the trochlea. He believed that suture gave perfectly adequate fixation when the arm was supported in a comfortable degree of flexion.

Clinical meeting—On May 10 a clinical meeting was held at the Robert Jones and Agnes Hunt Orthopaedic Hospital, Oswestry. Over 200 patients had been assembled to demonstrate a host of lesions and their treatment. In the afternoon many tributes were paid to the memory of Sir Robert Jones, not least in the delightful reminiscences of *Mr Billy Burton*, senior transport driver at the hospital. *Mr S. A. S. Malkin* (Nottingham) showed a film of Sir Robert at work at St Thomas's Hospital, obtained through the good offices of *Mr R. W. Furlong* and subsequently presented to the Association. Clinical demonstrations were then given by members of the staff. *Mr F. G. Allan* spoke on " Problems of leg lengthening," *Mr H. Osmond-Clarke*, *Mr Robert Roaf* and *Mr F. G. Allan* on " Scoliosis," and *Mr W. A. Law* on " Late results of cup arthroplasty."

Osteochondritis of the thoracic spine (Scheuermann's disease), sacroiliitis condensans, and ankylosing spondylitis—*Sir Reginald Watson-Jones* showed patients and radiographs to support his view, which was not intended to be more than a preliminary observation based on about four years' study, that

osteochondritis of the thoracic spine hitherto known as Scheuermann's disease was in fact a variant of ankylosing spondylitis easily cured by irradiation in low dosage of about 800 roentgen units without need for prolonged immobilisation in plaster beds, plaster jackets or spinal supports. He pointed out that whereas ankylosing spondylitis with sacroiliitis as the first evidence of it had become increasingly recognised in boys aged eighteen to twenty years, it was now being recognised in girls aged twenty-five to thirty-five years; and suggested that radiographic examination of the sacro-iliac joints in young adolescents with " Scheuermann's osteochondritis of the thoracic spine " would very commonly show a similar sacro-iliac disorder, and that this was one more variant of a hormonal disorder which could be cured quite simply by carefully coned and low-dose irradiation.

On May 11 *Mr H. J. Seddon* (London) took the chair to introduce three members of the Medical Research Council committee on vaccination in poliomyelitis.

Field studies in vaccination against poliomyelitis—*Professor C. H. Harris* (Sheffield) called attention to the previous lack of any controlled trial before the general introduction—against diseases such as typhoid and diphtheria—of various public health measures which have now become standard practice. There were those who argued against any preliminary clinical trial in the case of poliomyelitis vaccine, on the grounds that proof of the vaccine's antigenicity in monkeys was sufficient evidence of its being beneficial, that large-scale experiment on children was morally unjustifiable, and that in any case the American trial would soon produce the evidence required. The latter, however, left certain questions unanswered, and the disaster with the Cutter vaccine compelled, in the British view, some modification of the vaccine, so that the American results were no longer applicable. Therefore the British vaccine was introduced in such a way that its effect could be measured. He then explained the method of selection of children to be inoculated. Because of the low attack rate, which diminishes with increasing age even though the mortality among cases of the disease increases, very large numbers are required to produce significant results, yet the children selected for inoculation must cover a wide range of ages to exclude such possibilities as that the vaccine might be effective only in older children who had already acquired some natural immunity. An appeal for volunteers yielded 30 per cent of the 6,000,000 children aged one to nine years. The fact that the vaccine was in such short supply enabled controls, as far as possible free from bias, to be obtained from volunteers who could not be inoculated, a problem which would otherwise have been extremely difficult for a number of reasons. Selection of cases for inoculation was on the basis of month of birth, those aged five to nine born in November and younger children born in November and March being offered the vaccine; these months were chosen because the numbers born in them fitted the quantity of vaccine available. Two inoculations were given to 178,161 children, and 32,379 received one dose only. The remaining 1,669,553 registered children formed the control group; to give an inert inoculation to this group—the ideal method—was not practicable.

The next problem was to ensure that all cases of poliomyelitis in both groups were reported to the Medical Research Council Statistical Unit. With a notifiable disease usually treated in hospital this was not unduly difficult. The occurrence of missed cases, more likely in younger children and conceivably among inoculated children due to modification of the disease, had to be accepted. All cases, both paralytic and non-paralytic, notified within the ages one to nine, were reported to the Unit, and for all registered children among them clinical reports were requested three weeks and three to five months later. From these reports it is possible to answer the question whether or not inoculation as given reduces the subsequent incidence of paralytic poliomyelitis. They provide no answer, however, to the question whether inoculation influences infection by the virus—that is, reduces its ability to infect the gut, a matter only to be settled by laboratory tests. The latter are also essential in the diagnosis of non-paralytic poliomyelitis, where the clinical picture of aseptic meningitis can be caused by many viruses other than poliomyelitis. A third function of the laboratory is to type the virus involved; Type I was found in more than 80 per cent of cases, thus providing an adequate field test of the effectiveness of the Type I strain used in the British vaccine, a strain quite different from that used in the Salk vaccine.

Professor Harris then reviewed the possibility of reactions and other adverse effects. The incidence of immediate local reaction was very low because the protein content of the vaccine was small, and similarly sensitisation to antibiotics was very unlikely. Immunisation to monkey protein was possible, but tests for antibodies arising from the presence of Rh antigen and for nephritis from monkey kidney material had all proved negative. The exclusion of live virus by stringent safety tests would prevent direct infection; the possibility that latent poliomyelitis in the child might be provoked by the injection was also under consideration.

Preparation and testing of the vaccine—*Dr W. L. M. Perry* (London) explained that the basic process was to incubate the virus for three days in a medium containing a tissue culture of renal cortical cells from the rhesus monkey; in this process the renal cells were destroyed. After filtration the

resulting suspension of living virus was treated with formalin for fourteen days. An excellent colour film from the Glaxo Laboratories was shown to illustrate the extreme complexity of the measures necessary to carry this basically simple process to completion.

Dr Perry gave a brief account of the disaster in the United States with the Cutter vaccine. Virus was isolated in about half of the 200 cases of poliomyelitis that resulted, and of those isolations all but one were of the Type I Mahoney strain. The presence of living virus in the offending batch of vaccine could however only be demonstrated easily in tests on cortisone-treated monkeys, most of the tests in tissue cultures of renal cells proving negative. Furthermore the monkey tests demonstrated the presence in the vaccine of living virus of all three types. This discrepancy has never been explained and is the main reason for the rejection of the Mahoney strain of Type I for use in the British vaccine.

In testing our vaccine tests are carried out both by the makers and by the Medical Research Council. In each laboratory nearly two litres from each batch is cultured and subcultured over a five-week period, and tests are also carried out by intracerebral, intraspinal and intramuscular injection on both rhesus and cynomolgus monkeys, the latter being even more sensitive than the former. Tests for antigenicity are made in monkeys after three injections at weekly intervals, the content of antibody in the serum being estimated by its capacity to neutralise living virus added to tissue cultures.

Live virus vaccination—*Professor G. W. A. Dick* (Belfast) stressed that whereas Type I virus was responsible for most cases of paralysis, and Types II and III much more rarely, for most people infection was a subclinical alimentary infection. The antibodies produced by each type of infection were specific. The object of work with live viruses had been to reproduce the immunity associated with natural infections, with the added advantage that the vaccine could be taken by mouth.

The first strain studied was an attenuated Type II virus originally used successfully by Koprowski. This strain was considered suitable because it never produces paralysis when inoculated intracerebrally in monkeys and only rarely when injected into the spinal cord. Furthermore it is non-cytopathogenic—that is, it does not destroy the cells in monkey kidney tissue culture, and it was reported to be excreted only occasionally and in small amounts by vaccinated individuals. It was therefore tried on 190 children, mostly from medical or university families, and the satisfactory proportion of 77 per cent developed Type II antibodies. Unfortunately it was found that the virus was excreted by all subjects who developed antibody, and in large quantities in some individuals. Furthermore, the excreted virus was both cytopathogenic, and paralytogenic in inoculated monkeys. Because of the potential danger of this strain further study of it was abandoned.

Trials of an attenuated Type I strain (SM virus) avirulent for monkeys but known to be excreted in large quantities by vaccinated individuals, were made to discover whether it too would change character on gut multiplication and whether it was likely to be transmitted within a normal family. The answer to both questions was affirmative, and SM virus was therefore also abandoned as unsuitable for wide scale use, for which purpose Professor Dick and his colleagues considered 1) that the virus should remain avirulent on passage through the gut, and 2) that the virus should not be transmissible from vaccinated to non-vaccinated individuals. At the moment it appeared that no such avirulent strain was available.

It might be possible to develop a strain excreted in such small amount after vaccination that spread was unlikely; the immunity produced would, however, be unlikely to be good. Also, though a virulent infection might produce long-lasting immunity, infection with milder and with avirulent strains probably produced lower antibody levels which fell away subsequently. Naturally acquired immunity in many parts of the world probably depended on periodic reinforcement by reinfection. There was at present no evidence that three injections of a good Salk-like vaccine produced an immunity inferior to that produced by attenuated strains, and he thought that one line of future research should be directed towards developing a purified dead virus preparation of high antigenicity rather than towards the present types of live virus vaccines.

Answering questions, Professor Dick said that the labelling of virus by radioactive tracers, while not necessarily impossible, was impracticable at present. Assessment of the risk that passage through the gut would cause the virus to become virulent could be made on monkeys, but only if chimpanzees were used.

Calcinosis circumscripta—a local and metabolic study—*Dr R. W. Bailey* (Los Angeles) said that calcinosis, first described by Webber (1878), could be divided into two groups, calcinosis circumscripta—usually seen in women and associated with scleroderma, sclerodactyly and Raynaud's syndrome—and calcinosis universalis, often an affection of children. Abnormal calcium and phosphorus metabolism, local failure of circulation and some endocrine upset associated with the menopause had all been considered in the etiology, but without clear-cut evidence. He reported a case in a woman of sixty-three, whose hands and elbows were chiefly affected. She was in slightly positive calcium balance; phosphatase and other laboratory studies were normal. Biopsy showed that fascial planes

and muscles were affected, the latter showing intramuscular collagenous degeneration in biopsy specimens with thinning of the overlying skin. He described the action of chelating substances, and had tried the effect of one of these, versene, in this patient in the hope that it might bind with the calcium in the deposits and help disrupt them. Although increased calcium output in the urine followed there was no clinical change, nor reduction in the calcium percentage in the ashed deposits. Versene had caused auricular fibrillation which ceased on withdrawal of the drug. He concluded that calcinosis was not a general metabolic disease but a local change. There was no evidence of preferential removal of calcium from the deposits. He postulated that in these cases something was lost from the cells which normally protected them from calcification and thought that future work should aim at further understanding of histochemistry and cell metabolism. *Mr H. J. Seddon* (London) commented that calcification of similar type was seen in cases of Volkmann's ischaemic contracture.

Cineradiography of the normal cervical spine—*Dr J. W. Fielding* (New York) showed an excellent film. By photographing an image-intensifying screen he had been able to demonstrate with the greatest clarity the movements that occur at various levels, those in the upper cervical spine being of particular interest.

Cancellous strip bone grafting—*Mr J. N. Wilson* (London) had analysed twenty-two cases of cancellous strip grafting for delayed union of fractures of long bones and he described the steps of the operation. It was essentially a procedure applicable to delayed union but could be adapted to the treatment of non-union. Grafts were cut from the crest of the ilium in all cases and laid around the fracture site in a prepared bed. This produced a bypass type of graft resulting in union which resembled the " lead pipe join " of a plumber. Whenever possible the pseudarthrosis was left undisturbed. If the fracture had to be taken to pieces, then internal fixation was advised to supplement the grafting. The grafts had never been employed as an internal splint. When a plate had been used previously it could be left in situ and the grafts laid around the site of plating. External splinting was essential in all cases. There were two cases of failure, both of which could be explained by errors of technique. Several fractures had been grafted in spite of infection and, although there was partial sequestration, most of the grafts survived and the fractures united. The advantages of the method could be summarised as follows: 1) it was a simple and less traumatic procedure than established methods of grafting, although skin suture might be difficult; 2) it could be carried out successfully in the face of infection; and 3) the grafts were not used for internal fixation and were not liable to fracture. Also there was no danger of fracture through the donor site. *Mr John Charnley* (Manchester) endorsed the procedure and considered it one of the great advances in surgery. He used it in the case of the tibia if the fracture was not firm at three months, only grafting the inner and outer aspects and not posteriorly. Skin closure was sometimes difficult and he advised soft-part stripping and stretching with the fingers. *Mr Robert Roaf* (Oswestry) thought the method applicable to cases of non-union if of the type which might be considered suitable for drilling. He never used metallic internal fixation in these cases, especially as many were old compound fractures. He had had no trouble from using skin flaps, which made suture easy, and recommended lifting a shaving of bone with the skin flap.

The etiology of idiopathic pes cavus—*Mr J. N. Aston* (London) had reviewed 755 cases of idiopathic pes cavus from hospital records. The information thus obtained was limited, and there was a bias in favour of the severer case, whatever aspect was considered.

His findings were that pes cavus did not often present under the age of five, reached a peak between nine and thirteen, and then fell off again. Males predominated over females throughout; male cases also tended to present earlier and were rather more severe in the younger patients. Nevertheless, when progressive cases were considered, this early male preponderance was not seen. The progressive cases did not seem to present any younger than the average.

Six clinical features were next reviewed—varus of the forefoot, varus of the heel, clawing of the toes, dropping of the first metatarsal head, valgus of either heel or forefoot, and asymmetry between the two feet. It was found that those with varus of the heel did badly, those with valgus did well. Clawing was the commonest individual feature. Patients with clawing and dropping of the first metatarsal head did not do as well as those in whom these were not marked—possibly, in the case of claw toes, because they presented later. Forefoot varus and asymmetry between the feet seemed to make no difference to the outcome.

The association of pes cavus with spina bifida occulta was discussed, and some relationship between them sounded probable. A laminar defect was found in fifty-two out of 104 patients radiographed, against about 18 per cent in normal people. There did not seem to be any association with a bad prognosis.

The surgery of fixed claw toes—*Mr A. W. Fowler* (Bridgend) described an operation which had been done on twenty patients with irreversible claw toes. A transverse dorsal incision was made just proximal to the webs of the toes, dividing extensor tendons and exposing the proximal phalanges.

The proximal half of the proximal phalanges was removed and the metatarsal heads were exposed. These were trimmed so that there was a level surface underneath and an even line anteriorly. After closure of the dorsal skin incision an ellipse of skin was removed from the plantar surface of the foot behind the metatarsal heads. Sufficient skin was removed to pull the metatarsal weight-bearing pad back into relationship with the reconstituted metatarsal heads. A plaster toe cap was worn for six to twelve weeks. All the patients had gained very great relief from the operation. They were particularly grateful for relief of pain beneath the metatarsal heads, and some of the most satisfied patients were those with old rheumatoid arthritis.

SPEECHES AT THE DINNER OF THE ROBERT JONES CENTENARY MEETING OF THE BRITISH ORTHOPAEDIC ASSOCIATION IN OSWESTRY, MAY 1957

From Lady Probyn Jones

Daughter-in-law of Sir Robert Jones

Mr President, my Lord, Ladies and Gentlemen—Up till to-night I have been able to avoid speaking in public. I even got out of it when my husband was standing for Parliament. Then it became necessary for me to change my politics overnight! But this evening I am strengthened by the knowledge that surgeons must be used to very nervous people and disarmed by the photograph in front of me as I always am over things concerning my beloved Uncle Bob.

I will tell you now one or two instances to illustrate those characteristics which endeared him to his family. My first recollection of him is of being taken from my cot, rolled in a blanket and taken screaming downstairs and put upon his knee. I would let no one near me—permit no one to touch—even look at my injured leg. I remember vividly his *blue*, *blue* eyes and his beaming smile. We were close friends at once. My mother told me he had fixed my leg, and I had not even noticed. It was then that he christened my doll, Rhubarb Elsie.

There are many of these Major-General stories, and some have been told by my brother-in-law in his book on the life of Robert Jones, but I would like to tell you of the conversation I overheard between my uncle and my husband—" Father, If we meet anyone we know when we are out, as you are in uniform, do please try to remember not to raise your hat! "

I used to help my uncle sometimes to entertain his guests at dinner. He would telephone me, and one day I answered the telephone and he said: " Darling, a very brilliant young student is coming here to-night—will you help me? " I said, " Of course, but what is his name? " He said " It is Reginald Watson-Jones but I think you call him David." On one other occasion I was able to help in this way. My uncle loved his Grand National night dinner party. But on this particular night things looked like flagging after the meal when we had moved to the drawing room. My uncle walked right over to me and said: " Eileen, do go to the piano and sing—I think it will help to start up the conversation." This is exactly what you must be longing to do now—so I will end by thanking you again for the privilege I have been given in asking me here to-night.

From Mrs Freda McMurray

Formerly Miss Evershed—secretary to Robert Jones for ten years

Mr President, my Lord, Ladies and Gentlemen—I feel it is a great honour to be asked to speak at this dinner to-night, though like Lady Probyn Jones I was struck dumb with horror and dismay when I received Mr Wiles's letter, but as Mr Osmond-Clarke has told you I was Sir Robert Jones's secretary for the last ten years of his life; and very busy, happy years they were too.

Sir Robert was such a tremendously hard worker, and inspired his staff with the same zeal and enthusiasm. His routine day was to start operating at 8.30 a.m. and then followed a spate of patients at Nelson Street, which went on until the late afternoon. On Thursday afternoons he went up to London where he followed the same routine on Fridays. At the week-end he frequently relaxed at his daughter's home in North Wales, where he enjoyed a day's shooting during the shooting season; and once a month he operated at the Shropshire Orthopaedic Hospital on a Saturday. When he was busy writing, and he wrote a great deal, he would return to Liverpool for the week-end, and I would go up to Belvidere Road so that he might dictate to me.

Perhaps some of you do not know that it was Sir Robert who brought the first x-ray apparatus into this country in 1895 or 1896 and he and Mr Thurstan Holland experimented with it. It was these experiments which led to Mr Holland's taking up radiology. I understand that the first x-ray taken was of a boy's wrist with a Colles's fracture and it was given an exposure of one and a half hours!

In those earlier days when Sir Robert was running that famous Sunday morning Clinic, when people came to him from far and wide, he still found time to relax when work was over, and frequently boxing bouts took place in the studio. He engaged two boxing professionals, one named Meadows, and I think the other one was James, to come and instruct him, the young doctors assisting him in the art of boxing.

It does not need me to tell you of his generosity and kindness, and you all know that he inspired affection in all those who came into contact with him.

One thing that always struck me was his insistence on the team spirit, as he believed that the best work was done only when the team worked in harmony. He did not believe in the lone wolf who went his own way for his own ends.

Sir Robert had so many dear and lovable ways, and he was always very funny about money. He never had a cheque book as I kept this in the office, and when I thought he might need some money I cashed a cheque and gave him the cash. Sometimes when I would go into the studio his wallet was laid out in a prominent position so that I might see that it was empty, and in an aggrieved voice he would say to whoever was there: "I work very hard, but no one ever thinks I want any money; I have to ask Miss Evershed for my pocket money."

I think you all know that Sir Robert had a young surgeon assisting him at Nelson Street, and after about a year's training the young man would go on to pursue the practice of orthopaedic surgery on his own. My husband was the last of these young men, and he remained with Sir Robert until the end, but what you perhaps do not know is that besides being his assistant he was also his hairdresser.

Sir Robert would be dictating to me in the studio when my husband would come in, walk round Sir Robert and say, "Sir, your hair wants cutting." "No, no, no, McMurray, it is perfectly all right as it is." "No, Sir, it is far too long, it is nearly over the top of your collar." A sheet would then be spread on the floor, a chair placed in the centre, and, still protesting, Sir Robert would be divested of his coat, put into a white coat and wrapped in a towel and be led to the chair. The hair cutting would then begin and the dictation continue at full speed, with frequent shrieks of "McMurray, you are cutting my ears, I am sure you have done enough." My husband would go stolidly on until the end, when a mirror would be held up for Sir Robert to see the result. He was always very satisfied and would give my husband a sixpence tip which promptly went into the Heswall collecting box!

On looking back one thinks of Sir Robert's boyishness, for he seemed to personify perpetual youth, so that it is impossible to think of him growing old, and I feel that that youthful spirit must still be with him to-day.

From PHILIP WILES, M.S., F.R.C.S.

President of the British Orthopaedic Association

It is a platitude to say that Robert Jones is the father of orthopaedics, but it happens to be a fact, and I am his orthopaedic grandson. Thus it falls on one of the third generation to have the privilege of presiding over the Association that he founded, and the pleasure of proposing this toast. There would be no happier subject about which to speak, but it is not so easy for one who knew him as slightly as I did, especially in the presence of a few, and alas they are now very few, who knew him intimately.

A hundred years is a long time and I want just to remind you of what the world was like then. The British Empire was still expanding and 1857, the year of his birth, was that of the Indian Mutiny when a part of the gigantic private army of the East India Company rebelled against their conditions of employment. He grew up during the period when the Empire was consolidating and had become the paramount power in the world. But he matured in the great days of Liberalism; that was the time when men came to realise that they had not only rights but also duties—duties to their fellow-countrymen and duties to their fellow humans everywhere. That was the atmosphere of the time, an atmosphere that Robert Jones, one of the great men of the times, himself helped to create. For it was in the field of kindness and of love that he was supreme. Witness his work among the poor of Liverpool; his care of the navigators constructing the ship canal to Manchester; the inspiration that led him to create the first service for the care of crippled children; his triumph in the first war when he was directly responsible for saving untold thousands of lives; and, even more important, succeeded in making the Government appreciate that it had a duty to the soldiers mutilated fighting its battles.

I don't know whether Robert Jones was precocious, but he certainly made a quick getaway with his career. At the age of twenty-four, only three years after he qualified, he was appointed assistant surgeon to the Stanley Hospital, Liverpool, and at twenty-nine he became full surgeon and Dean at the Southern Hospital. This early start, together with an immense capacity for work, combined to give him a unique experience of orthopaedic problems at a very young age. His speed of work must have been terrific. It is said that an ordinary afternoon's list in the theatre consisted of twenty to thirty operations, many of them major ones. Minor operations such as tenotomy were performed in the clinics and consulting rooms, often without anaesthesia. There is a story that Robert Jones was once reducing a dislocated shoulder in a nursing-home, of course without anaesthesia. It had been out for some days and was rather difficult, but finally he succeeded in getting it back. He chanced to leave the room at the same time as an obstetrician from next door. The latter started to complain about the noise that Robert Jones's patient had made, pointing out that he had just

delivered a lady of a baby and she had not made a sound. "Ah!" came the characteristic reply, "but you just try putting it back again!"

A little while ago a student in my clinic was saying how easy it must have been to become famous if only one had been born in those days when the diseases and ideas with which we are now so familiar were still waiting to be discovered. I suppose he imagined them like great fruits hanging from the tree of knowledge waiting to be knocked off by the first passer-by with the energy to throw a stone. But it is only after long and tender cultivation that the tree can be persuaded to yield its fruit. Even then it is given only to a few great men to make new discoveries and propagate new ideas. Robert Jones was one of these great men. Look at the genius of Baschurch where, together with Agnes Hunt, he created the service and founded the hospital which we visited to-day and which remains as his living memorial. He started there a new era in the treatment of disease, and his ideas were immediately adopted by progressive surgeons everywhere. Even to-day he is emulated. There is a good example in Jamaica where at this moment an orthopaedic great-grandson, John Golding, is creating a new service and meeting the same difficulties that must have confronted Robert Jones. Before he can hold a clinic or use a workshop he has to build the buildings and train the staff; and before he can build the buildings he has to raise the money; and to transport the children he has often to use his own car with himself as chauffeur. For anyone imbued with the spirit of Robert Jones there are still ample opportunities in many parts of the world.

The great war, that is the first world war, found Robert Jones at the height of his power, and his work at that time was destined to have the greatest influence on the future of orthopaedic surgery. By the end of the war he had collected around him a great family of eighty surgeons, British and American, who were destined to become the second generation of orthopaedic surgeons. Robert Jones in the Army must have been great! He could not abide assumed and unnatural dignity in any form, and he never came to understand the attitude with which a soldier regards his uniform. Even in his young days as a member of the Corps of Volunteers he thought there was nothing very wrong, when the trousers of his uniform were split, in turning up on parade for a general inspection wearing civilian trousers. And I would have given anything to have seen him dressed as a Major-General in the full glory of his red tabs walking down Whitehall on the way to the War Office with an umbrella up! When someone remonstrated with him gently he replied, "But why not? It's raining, isn't it?"

Robert Jones achieved in his life probably more than any other surgeon before or since. He succeeded not only because of his great experience and wisdom, but equally because of an unflagging humanity and an indefinable charm of manner. It has been said that his arrival gave the same impression that the sun gives when it comes out on a dull day. What better epitaph can a man have:—*he was like the sun when it comes out on a dull day.*

I am now going to give you a toast, but that toast is *not* to the memory of Robert Jones. Rise and drink to the ever living spirit of Robert Jones!

OBSERVATIONS AT THE OPENING CEREMONY OF THE CLINICAL MEETING IN OSWESTRY AT THE ROBERT JONES CENTENARY MEETING OF THE BRITISH ORTHOPAEDIC ASSOCIATION

From WILLIAM BURTON

In charge of the Motor Transport at Baschurch and then at Oswestry for nearly fifty years

My earliest recollection of Sir Robert was in 1910 when I was then a schoolboy at Baschurch. I remember Dr Jones's visits (as we *then* knew him)—he arrived on occasional Sundays by horse and trap at the house known as Baschurch Surgical Home. This house, with two open-air sheds in the garden, accommodated about twenty crippled children. It was in a room of this house where Sir Robert first performed operations at Baschurch. In those days it was a familiar sight to see one or two spinal carriages, a pony and dray loaded with half a dozen children and Sister Hunt aboard, on its way to Baschurch Station to board a train to Liverpool. They were met at Liverpool, pushed through the streets in carriages and handcarts to the Southern Hospital to Mr Robert Jones's out-patient clinic. The children left on a previous visit were brought back on this journey probably in exchange for some left behind that day.

From these early days my recollections cease, until I became a member of the staff in April 1919 after being demobbed from the Army. Later that year Sir Robert's visits to Baschurch became more frequent, although he was still on military service; it was part of my duties to meet him at Shrewsbury station and drive him to Baschurch for his week-end visits in the only car we possessed—a 1914 Ford convertible. He would sometimes arrive dressed in the uniform of a Major-General R.A.M.C. I had a feeling on those occasions that the old Ford was not quite befitting to the red-tabbed uniform he wore, but I have pleasant recollections of the friendly conversations on these journeys.

On his monthly visits the whole staff were keyed up in case anything was wrong and we did not relax until his departure on Sunday evening, although of course we all looked upon him with great respect—he always had a smile and a word for any member of the staff. I remember when an Albee was a rare operation at Baschurch—word would go round that Sir Robert was performing this operation and you could see on the faces of the staff—and by their conversation—that everyone had an interest in this wonderful operation. Stand-to's in those days would be regarded to-day as something in the nature of a pantomime—patients would be lined up in spinal carriages and as many as four small children would be set out on a single bed for examination. On one occasion Sir Robert was heard to ask Sister Hunt—" Are you sure there are no more *under* the bed?"

About 1919–20 when Sir Robert returned from military duties the visits to Liverpool were resumed. By that time the Baschurch Home had been presented with a Ford van, convertible to an ambulance. By sliding in a few boards between the stretcher platforms we were able to transport half a dozen children on frames or bed splints, lying head to foot alternately, in comparative comfort direct to Sir Robert's consulting rooms in Nelson Street. We would set off about 8.30 a.m. complete with sandwiches and lemonade, bottles and bedpans, and with Sister Hunt in attendance arrive at Nelson Street about midday, after a few stops on the way and the inevitable wait at Birkenhead in the queue for the ferry. We aimed to leave Liverpool at about 4 o'clock, but this did not always work out and on occasions had to wait for two hours in the ferry queue on the Liverpool side. This did not improve the spirits of the party and it was generally about 8 p.m. before we arrived back at Baschurch.

With the return of peace—and the regular week-end visits of Mr Aitken, Mr Naughton Dunn and Mr Girdlestone—these journeys to Liverpool ceased. About this time Sister Hunt would often drive herself and a doctor to visit the After-Care Centres. These journeys were rarely made without incident. Traffic was light on the road, but I have known drivers of horse-drawn vehicles, and cyclists, going into the ditch or disappearing hastily up a side road to allow her plenty of road space. She delighted in running as close as possible to the toes of policemen on point duty in Shrewsbury; they all knew her of course and so did the Chief Constable. I would like to mention one of the many incidents that occurred when she was driving. About 1923 Sister Hunt and a doctor went to Cumberland and Westmorland to help organise the opening of After-Care Centres there. I drove on the first stage to Lancaster. After lunch there she decided that she would drive on to Carlisle. The doctor pleaded with her to let me continue at the wheel, but to no avail. We got up Shap all right, but on a long descent to Penrith I could see in the distance an animal moving about the road. I thought—" If she doesn't slack off or do something, somebody is for it here." As we got nearer I could see that the animal was a pig—she tried to swerve around the pig and he had the same idea and direction for we carried him into the ditch and out again. When she pulled up after several yards I got out and underneath the car found the pig entangled in the under-carriage. Sister Hunt very calmly said, " Billie, I smell bacon." That was not surprising as the pig was firmly stuck against the hot exhaust box.

To finish I would like to say how proud I feel to have been given this opportunity of telling you a few recollections of Sir Robert and Dame Agnes of nearly forty years ago. After this long period my memories of Sir Robert and Dame Agnes are happy ones and will always remain with me.

Election of Fellows, Members and Associates

At a Meeting on May 9 the following were elected.

Fellows—G. L. W. Bonney (London), J. M. Fitton (Leeds), J. S. R. Golding (Jamaica), R. C. J. Hill (Durban), G. C. Lloyd-Roberts (London), A. McDougall (Glasgow), W.R. D. Mitchell (Northampton), E. J. Nangle (Salisbury, Southern Rhodesia), B. Polonsky (Johannesburg), G. K. Rose (Oswestry), W. T. Ross (Johannesburg), W. H. M. Smith (Doncaster), F. B. Thomas (Hereford).

Members—N. J. Blockey (Glasgow), E. G. Herzog (Sheffield), N. E. James (Grimsby), R. H. Maudsley (Windsor), R. Merryweather (Gloucester), R. L. Rees (Cardiff), J. H. Saunders (Lower Hutt, New Zealand), E. Shephard (Maidstone), W. Sillar (Glasgow), F. W. Taylor (Sheffield), A. G. M. Watt (Glasgow), W. F. White (Glasgow).

Associates—V. P. Amato (Sliema, Malta), H. Carrier (Chicoutemi, Canada), R. S. Cowie (London), D. K. Evans (London), L. W. Godfrey (Cambridge), J. M. Lancaster (Newcastle upon Tyne), C. R. F. McDowell (Dublin), R. O. Murray (London), H. K. S. Murthy (Warrington), H. E. Piatigorsky (London), P. A. Ring (London), F. W. Shea (Shrewsbury).

ARRANGEMENTS FOR FUTURE MEETINGS OF THE BRITISH ORTHOPAEDIC ASSOCIATION

The spring meeting will be held jointly with the American Orthopaedic Association in Washington from May 11–16, 1958. The autumn meeting will take place at Oxford on October 2, 3 and 4, 1958.

ROBERT JONES GOLF COMPETITION

The Annual Competition for the Robert Jones Cup was played this year at Hawkstone Park Golf Club in Shropshire during the Spring Meeting of the British Orthopaedic Association.

The competition was graced by good weather and the course was in excellent condition. All competitors were unanimous in praising the picturesque surroundings of this delightful golf course. The play reached its usual high standard and the competition was won by Mr R. H. Maudsley with a score of 78−6=72. He won by one stroke from Mr C. C. M. Murray and Mr A. E. Bremner, who tied for the position of runner-up.

After the competition the golfing members who had taken part enjoyed an excellent dinner at the Hawkstone Park Hotel under the direction of Mr Gordon Irwin, the retiring Captain. Mr W. H. Maitland Smith of Doncaster was elected Golf Captain for the next two years.

As the Spring Meeting of 1958 will be held in Washington the next competition for the Robert Jones Cup will probably be at the Autumn Meeting at Oxford in October of 1958.

ROBERT JONES CENTENARY SERVICE IN LIVERPOOL CATHEDRAL, JUNE 1957

A commemorative Service was held on June 28, 1957 in Liverpool Cathedral on the centenary of the birth of Robert Jones where his ashes are laid beneath the wonderful stained-glass window which is dedicated to "Service." Many surgeons from Britain and the Commonwealth attended the Service.

ROBERT JONES DINING CLUB

In the Centenary Year of Robert Jones's birth, it was fitting that the Robert Jones Dining Club should hold its dinner—the twenty-ninth—in Liverpool, his home and the scene of his greatest labours. His successors there made this a worthy occasion that would have gladdened the heart of R. J.

The attending members, who included Dr R. I. Harris, were given lunch at the Royal Southern Hospital—where Robert Jones worked for twenty-nine years—by Professor Bryan McFarland, Clerk of the Club, and Mr Norman Roberts. Afterwards, at the Liverpool Medical Institution, many historic relics were on view in the Robert Jones Library, and a spirited meeting was held by the Liverpool Orthopaedic Circle. From there a pilgrimage was made to what had been 11 Nelson Street, and thence to the Cathedral, where the Dean, the Very Reverend F. W. Dillistone, conducted a brief and moving service at the stone that marks the last resting place.

In the evening the dinner, at the Adelphi, was held in a spirit of thankfulness, and as the clock struck nine the silent toast was drunk.

SCIENTIFIC SOCIETIES AND REGIONAL ORTHOPAEDIC CLUBS

THE BONE AND TOOTH SOCIETY

The Bone and Tooth Society held a symposium on " Tissue culture and induction of bone and connective tissues " at the Institute of Orthopaedics, London, on October 4, 1956, with Mr I. R. H. Kramer in the Chair.

Bone and cartilage induction in the rabbit, rat and guinea-pig—*Dr J. B. Bridges* and *Professor J. J. Pritchard* (Belfast) considered that the possible role of bone induction in bone growth and repair could be better assessed if a reproducible method for eliciting heterotopic bone and cartilage formation could be devised. Two procedures were chosen. 1) *Implantation of alcohol-fixed tissues beneath the kidney capsule of the rabbit*—Alcohol-killed hypertrophic cartilage, whether from fracture callus or growing epiphyses, regularly underwent bony replacement, leading to the formation of an ossicle filled with haemopoietic marrow. Alcohol-fixed skeletal muscle first induced hypertrophic cartilage, which secondarily underwent bony replacement and reorganisation into a marrow-filled ossicle. 2) *Ligation of the renal vessels in the rabbit, rat or guinea-pig*—This led to extensive intramembranous bone formation in the kidneys of all three species. Ossification began nearest the surviving transitional epithelium of the ureteric pelvis. These results suggested three kinds of skeletal induction: *a*) formation of new bone replacing alcohol-killed hypertrophic cartilage; *b*) formation of hypertrophic cartilage around alcohol-killed muscle; *c*) formation of membrane bone adjacent to living transitional epithelium. Alcohol-killed hypertrophic cartilage still showed inducing activity after heating to 55 degrees for five minutes, and after prolonged treatment with 0·01 N HCl, but not after boiling or treatment with NaOH. Alcohol-killed homogenous hypertrophic cartilage was active, but not heterogenous tissue, hyaline and elastic cartilage, cortical bone or periosteum. Hypertrophic cartilage was induced less consistently by alcohol-killed urinary and alimentary tract tissues than by alcohol-killed muscle. Living urinary tract, however, produced bone without cartilage under the kidney capsule.

Tissue culture as a tool in dental research—*Lady Mellanby* and *Mr R. J. Holloway* (London) said that the growth of tooth germs *in vitro* had been used mainly to investigate problems of normal tooth development, but could also be applied to test the effect of various substances upon that development. If lower first molar germs were dissected from seventeen to eighteen day old embryo rats or mice and placed on clots made from cock plasma and chick embryo extract, they developed during fourteen days the equivalent of about six days *in vivo*. From an early bell stage normal cell layers and cusps differentiated and dentine matrix was laid down, but no enamel matrix or calcification had yet been seen. When vitamin A (1,350 i.u. per millilitre) was added to the medium, radical changes occurred in the explant. Growth rate was greatly reduced, and although cusps began to develop they eventually regressed. The ameloblasts became cubical or short columnar and most of the rest of the enamel organ atrophied. Differentiation in the cells of the dentine papilla also lagged. If the tooth germ was transferred after seven days to normal plasma without added vitamin A for a further seven days, considerable recovery occurred. Additional vitamin D in the medium, up to 700 i.u. per 100 millilitres, appeared to have no effect upon development.

Studies on the cultivation of embryonic chick bones in chemically defined media—*Dr J. D. Biggers* (London) said that embryonic chick femurs and tibias had been cultivated on chemically defined media consisting of a mixture of amino-acids, vitamins, coenzymes, nucleotides, salts and other substances. Medium 858 of Healy, Fisher and Parker (Proceedings of the Society for Experimental Biology and Medicine, 1955, **89,** 71) and modifications of it were used. The lens paper technique of Chen (Experimental Cell Research, 1954, **7,** 518) for the culture of organs was employed. The embryonic cartilaginous rudiments almost doubled their size during a culture period of six days provided adequate supplies of glucose were available, and their nitrogen content increased. The histology of the rudiments was well preserved although after six days' cultivation degenerative changes occurred. Biochemical studies on the medium showed that a considerable quantity of amino-acids was utilised. The fate of these amino-acids was only partly understood, but at least some were incorporated into the proteins of the rudiment. Preliminary studies on the " essential " amino-acids in the medium were reported. Experiments carried out in collaboration with Dr M. Webb on the synthesis of amino-acids from glucose were described. This synthesis had been revealed by the addition of 14C glucose to the medium and subsequent analysis of the proteins extracted from the rudiment.

Experimental embryology of tooth germs—*Mrs Shirley Hughes* (Cambridge) said that tooth germs deprived of their blood supply nerve supply, and their normal surroundings continued to develop in a normal manner under tissue culture conditions. The morphological and histological differentiation was as *in vivo*, although differentiation did not proceed quite as far. The problem of the inability of the functional ameloblasts to form enamel more often was as yet unsolved, as was that of the non-calcification of the newly formed dentine. It was suggested that the factors responsible for this

development were not present in the medium so far used in the cultivation of the tooth germs. The stage of development reached by teeth grafted on to chick chorio-allantois was considerably in advance of that previously achieved with tissue cultures. This advance was particularly related to the thick layers of prismatic enamel formed in the grafted teeth, and the calcification of both dentine and enamel. This difference in the general results of the two methods was presumably due to the presence of a circulation in the teeth grown on chick chorio-allantoic membrane.

SOUTH-EAST METROPOLITAN REGION ORTHOPAEDIC CLUB

A meeting was held at the War Memorial Hospital, Woolwich, on February 16, 1957, and attracted a large attendance. Cases were shown and discussed.

Osteitis pubis after inguinal herniorrhaphy—Three patients were demonstrated who suffered pain in the pubic region a few days or weeks after inguinal herniorrhaphy, progressing to inability to sit or to walk. All developed radiological changes of osteoporosis in the ischial and pubic rami and widening of the symphysis. In one case there was evidence of bone destruction in both ischial rami. These three patients had been operated upon by different surgeons and by different methods. One had a post-operative sinus for a short time through which several nylon sutures emerged, but in the others there was no evidence of wound infection. The symptom in each case subsided gradually with rest in bed, but it took several weeks to achieve comfort in walking and especially in sitting. The osteoporosis gradually resolved but some widening of the symphysis pubis was still visible in the later radiographs.

Mr A. D. LeVay had found no reference in the literature of this condition occurring after inguinal herniorrhaphy. Similar changes were well known after other pelvic operations such as prostatectomy and had also been described after normal childbirth. *Mr J. C. F. Lloyd Williamson*, despite a long experience of hernia operations, could not recall having seen such a condition and no one present could suggest how the osteitis arose.

Chronic synovitis of the knee—*Mr LeVay* showed a child with chronic synovitis of one knee joint and negative synovial biopsy, which had settled down after immobilisation and anti-tuberculous drug therapy. The Mantoux reaction was negative at the beginning of treatment but later became positive. *Mr W. G. France* questioned the reliability of synovial biopsy and was supported by *Mr D. Trevor* and *Mr E. A. Lindsey* in maintaining that inguinal gland biopsy was more reliable in the diagnosis of tuberculosis of the knee joint, because the tuberculous infection might not involve the whole synovial cavity and one could easily remove by biopsy a piece of unaffected synovial membrane. *Mr J. A. Cholmeley* agreed, but maintained that the difficulty could be overcome by inspecting the inside of the joint carefully at the biopsy operation and if necessary by removing a suspected area of synovial membrane through a second incision. Inguinal gland biospy, though probably the most reliable method for diagnosing tuberculosis, had the disadvantage of providing no helpful information if the synovitis was not tuberculous.

Excision of old tuberculous hip and leg shortening operation—*Mr LeVay* demonstrated an excellent result in a young woman with old tuberculosis of the right hip. After several years of conservative treatment as a child she had had a Brittain's arthrodesis in late adolescence in 1949 which led to bony fusion across the joint, but she was left with a stiff painful right knee joint and over four inches of shortening. Mr LeVay performed the following operations: 1) arthrodesis of the right knee; 2) excision of the remains of the head and neck of the right femur and of the Brittain bone graft with subtrochanteric angulation osteotomy of the right femur; and 3) subtrochanteric shortening of the left femur by excision of three inches of bone. A good range of movement had been restored at the right hip. *Mr F. G. St Clair Strange* voiced the opinion of those present in congratulating Mr LeVay most heartily on the excellent result.

Mr LeVay showed a second case of subtrochanteric femoral shortening by three inches in a patient with tuberculous arthritis of the opposite hip. *Mr J. I. P. James* agreed that the maximum shortening obtainable in the femur was three inches but pointed out that further shortening of a disproportionate limb could be obtained by shortening the tibia also. The general consensus of opinion favoured limb shortening rather than limb lengthening in cases of disproportion.

Bilateral osteochondritis dissecans of the knee—*Mr J. E. Buck* showed a girl with osteochondritis dissecans in both knees. Mr Buck had operated by Smillie's method on one side, replacing a large loose fragment of articular cartilage and retaining it by two pins. When these were removed six weeks later the cartilage was firmly fixed in place. *Mr D. Trevor* criticised the procedure, and maintained that more orthodox methods produced satisfactory results. He believed that refixing a loose fragment of cartilage made simple surgery more complicated and possessed no real advantages.

Spastic flat foot—*Mr Buck* showed a boy with unilateral spastic flat foot with an incomplete calcaneo-navicular bar shown radiographically. Relief of symptoms and signs lasting several weeks had been produced by injection of hydrocortisone with Xylocaine into the sinus tarsi as close as possible to the calcaneo-navicular joint.

Gap suture of extensor pollicis longus tendon—*Mr Buck* showed a patient treated by Trevor's method of bridging the gap with a nylon suture. *Mr Trevor*, having been critical of some of Mr Buck's other work, rose to voice his wholehearted support in this case. He argued that the results of this procedure were better than those obtained by tendon transplants and emphasised particularly the loss of power in the index finger which follows the use of extensor indicis tendon as a transplant. He also emphasised the need in this operation to trace the retracted proximal end of the extensor pollicis longus tendon by very carefully following the thread of granulation tissue which attaches it to its groove in the back of the radius.

Charnley compression for ununited tibial fracture—*Mr Buck* showed a patient in whom delayed union followed an infected compound fracture of the tibia. Despite some residual chronic bone infection the fracture had consolidated after six weeks in Charnley's compression clamp with Steinmann's pins above and below the fracture. He also showed radiographs of two other cases of delayed union successfully treated by the same method. *Mr R. C. F. Catterall* reported two transverse fractures treated by the immediate application of compression which had failed to promote union. Mr Buck argued that a layer of granulation tissue might be necessary between the bone ends for compression to have its effect in promoting union and *Mr R. H. Sewell* supported this view. He also had used the method successfully in cases of delayed union. *Mr J. T. Marcroft* pointed out that Charnley's method was developed for promoting rapid fusion of cancellous bone surfaces and *Mr J. I. P. James* emphasised that it was ineffective with compact bone surfaces in apposition. *Mr W. Laurence* reminded the meeting that an intact or united fibula could maintain a gap between the fractured tibial fragments which might necessitate the excision of a short length of fibula. The discussion was closed by *Mr H. L.-C. Wood* who questioned whether the success of this method did not depend essentially on the production of efficient immobilisation.

Dorsal interosseous nerve palsy—*Mr Buck* showed a patient with dorsal interosseous nerve palsy of gradual onset with no history of injury. *Mr E. Shephard* considered that this was an example of "neuralgic amyotrophy" described by Aldren Turner. *Mr Wood* had seen similar cases with spontaneous recovery and thought that the prognosis was good.

Partial paraplegia due to a bone tumour—*Mr G. B. Northcroft* showed a young woman who developed progressive paraplegia as the result of an osteogenic fibroma in the pedicle and body of the twelfth thoracic vertebra. Removal of the tumour was followed by gradual and ultimately complete recovery.

Diastematomyelia—*Mr Northcroft* also showed a case of this very rare condition in which a bony spur projects backwards from the posterior surface of a vertebral body. The cord may be split by the bony spur and the condition usually presents in infancy or childhood with signs of partial paraplegia. The lower thoracic or upper lumbar vertebrae may be affected. In the case demonstrated the patient was thirty-seven years old and the diagnosis rested on radiological findings during routine investigation for chronic low back pain without neurological disorder. A complete bony bar subdivided the neural canal into approximately equal halves at the level of the third lumbar vertebra, connecting the back of the vertebral body to the base of the spinous process. At operation it was found that the cord extended down to the fourth lumbar vertebra, having presumably been tethered by this bony bar, around which it was split into equal sections each ensheathed in normal membranes. Removal of the bony bar had been followed by uncomplicated recovery.

EAST ANGLIAN ORTHOPAEDIC CLUB

A meeting of the East Anglian Orthopaedic Club was held on March 30, 1957, at the West Suffolk General Hospital, Bury St Edmunds.

Epiphysial injuries—*Mr D. C. Tennant* demonstrated several epiphysial injuries which showed interesting points in their progress. These included: 1) A displaced head of radius with complete reduction but followed by cross union between radius and ulna leading to loss of pronation and supination. The child had a good functional result in spite of loss of pronation and some members thought it would be right to remove the bridge of bone. 2) Separated lower radial epiphysis with early epiphysial fusion. No deformity at the wrist had yet occurred but the child was being kept under observation. 3) Separated upper humeral epiphysis with considerable displacement. The way in which the humerus had remoulded itself to normal contours was the point of interest in this case.

Neoplasm in the thigh—*Mr J. Brennan* (introduced by Mr Martin) showed three cases of neoplasm in the thigh. 1) A fibrosarcoma beginning in the site of old osteomyelitis. Treatment was by high femoral amputation. 2) A leiomyosarcoma in the quadriceps treated by disarticulation through the hip joint. Death soon followed and at post-mortem examination a primary sarcoma of the uterus was found. 3) Liposarcoma of thigh, removed locally. There was recurrence six months afterwards.

Senile peripheral vascular failure—*Mr D. J. Martin* discussed senile peripheral vascular failure with treatment by use of intra-arterial insufflation of oxygen. Considerable discussion followed and some scepticism was expressed.

IRISH ORTHOPAEDIC CLUB

A meeting of the Irish Orthopaedic Club was held at the Royal Victoria Hospital, Belfast, on April 6, 1957.

Excision of coccyx—*Mr J. B. Pyper* reported the results of excision of the coccyx for coccydynia. In ten years the operation had been performed on thirty-six occasions. Twenty-eight of the patients had been traced. Twenty-five reported that they had either been cured or much improved, and none was made worse.

Research in osteology—*Professor J. J. Pritchard*, of the Department of Anatomy of Queen's University, told the members about the research work of his department. He went on to report the results of his work on the sources of calcium in fracture healing in rats, as studied with radioactive Calcium 45. Finally he introduced two of the members of his staff, Dr B. Bridges and Mr C. Nevin, who read short papers on " Heterotopic bone formation " and " Revascularisation of bone grafts " respectively.

Spinal fusion in tuberculosis—*Mr N. Martin* opened a discussion on spinal bone grafting in tuberculous spondylitis, with particular reference to anterior fusion by the transthoracic approach. He showed an excellent colour film of the operation.

Use of cortisone and hydrocortisone—*Mr J. Kyle* had used coritsone and hydrocortisone in a number of orthopaedic conditions. He reported good results in painful heel, tennis elbow, chondromalacia patellae and supraspinatus tendinitis.

Treatment of Perthes' disease—*Mr G. W. Baker* discussed the present trend to treat Perthes' disease by methods other than frame fixation. He had found fitting with a patten-ended caliper very satisfactory, and on a long term review more than worth while. He particularly demonstrated its value in those cases in which treatment began before there were demonstrable radiological changes in the metaphysis of the neck of the femur.

Intramedullary nailing of femoral fractures—*Mr R. I. Wilson* demonstrated a number of excellent results of intramedullary nailing of the femur in cases of multiple injuries. Primary union occurred in all of nineteen cases.

Rupture of musculo-tendinous cuff—*Mr R. J. Withers* discussed the treatment of ruptures of the musculo-tendinous cuff. He had found that injection of perabrodil into the subacromial bursa was often very useful in diagnosis. He demonstrated a number of cases in which an excellent result was obtained by operative repair.

AUSTRALIA

AUSTRALIAN ORTHOPAEDIC ASSOCIATION

The Annual General Meeting of the Australian Orthopaedic Association was held in Surfers Paradise, Queensland, from October 5–9, 1956. The following officers were elected: *president*—Mr A. R. Hamilton (Sydney); *past-president*—Mr T. King (Melbourne); *president elect*—Mr J. Colquhoun (Melbourne); *honorary secretary*—Mr F. H. McC. Callow (Sydney); *honorary treasurer*—Mr W. Hugh Smith (Sydney); *editorial secretary*—Mr W. Stening (Sydney); *executive committee*—Mr R. Hodgkinson (Sydney); Mr M. Maxwell (Sydney), Mr C. Hudson (Sydney) and Mr C. Langton (Sydney).

The Address by the President, Mr T. King (Melbourne), took the form of a colour film of the late Dr M. N. Smith-Petersen performing a cup arthroplasty for old septic arthritis of the hip.

Shoulder pain—A symposium was presented by *Mr C. Langton* (Sydney), *Mr E. West* (Adelaide) and *Mr E. Price* (Melbourne). *Mr Langton* dealt with shoulder pain from local causes only, recognising four groups: 1) capsulitis or " frozen shoulder "; 2) supraspinatus tendinitis and the painful arc syndrome; 3) supraspinatus tendon calcification; and 4) rotator cuff tears. He mentioned the diagnostic importance of radiographs in addition to recognised clinical signs and described cases of

osteogenic sarcoma of the head of the humerus presenting as frozen shoulder. Treatment of each condition was described. *Mr West* also discussed differential diagnosis and treatment both conservative and operative, and recalled also the difficulty at times of establishing whether the pain were due to local or referred causes. *Mr Price* reviewed fifty consecutive cases of shoulder pain at the Royal Melbourne Hospital between 1951–55. These were divided into: 1) acromio-clavicular lesions, 7; 2) supraspinatus injuries, 13; 3) acute calcinosis, 5; 4) chronic supraspinatus tendinitis, 6; 5) frozen shoulder, 10; 6) miscellaneous, 9. Only four of the patients were treated by operation. In the acute phase a frozen shoulder should not be manipulated, but rest and the maintenance of muscle tone by local measures were important. The acute pain in calcinosis was due to increased tension in the tendon and could be readily treated by aspiration with or without injection of hydrocortisone acetate at the same time. *Mr F. Callow* (Sydney) preferred a sling to an abduction frame in most cases, and discussed excision of the acromion in resistant cases and suture of the ruptured or partially ruptured tendon. *Mr J. Cloke* (Melbourne) asked the treatment of bicipital tendinitis. *Mr E. Spring* (Melbourne) mentioned the injection of hyaluronidase into the subacromial bursa. *Mr D. Parker* (Hobart) made a plea for the place of deep x-ray treatment. *Mr J. Lahz* (Brisbane) described three cases in which gout was first manifest in an acute painful shoulder, and discussed the differentiation between cervical spondylosis with referred pain and the " shoulder-hand " syndrome. *Mr R. Robinson* (Sydney) referred to the important psychological side, initiated by stress, and suggested the use of phenylbutazone (Butazolidin) in cases due to gout. *Mr N. Morgan* (Sydney) described a poor result from excision of the acromion and the bursa. *Mr R. Hodgkinson* (Sydney) mentioned villous synovitis as a cause of pain. *Mr G. Colvin* (Sydney) asked why cervical spondylosis and calcinosis of the supraspinatus tendon could be symptomless. *Mr H. Crawford* (Brisbane) favoured manipulation in chronic cases with only a certain degree of limitation. *Mr L. Teece* (Sydney) agreed. *Mr Langton*, in reply, described a biceps tendon test for bicipital tendinitis. He had found hydrocortisone disappointing on the whole. He thought that deep x-ray therapy probably relieved tension. He believed that associated cervical spondylosis was commoner than was appreciated. *Mr West* recalled that the cervical spine often showed radiological changes over the age of forty without symptoms.

Early bone grafting of fractures—*Mr J. Cloke* (Melbourne) limited his remarks to fractures of tibia and fibula. The large majority of patients were referred two or three weeks after injury when redisplacement had occurred in plaster. When simple screwing of a long oblique fracture was not possible he advocated either a sliding graft or an inlaid bone graft in preference to fixation with a metal plate. *Mr D. Sturrock* (Sydney) thought that early bone grafting was often indicated. He often used the iliac crest. *Mr D. Rowe* (Sydney) used metal plates with the addition of long cancellous slivers of bone. *Mr H. Crawford* (Brisbane) also recommended a plate plus a bone graft. *Mr D. Parker* (Hobart) used homogenous bone grafts for a time, but had now largely discarded these for autogenous iliac chips. *Mr L. Teece* (Sydney) thought that orthopaedic surgeons should avoid operation. *Mr F. Callow* (Sydney) recommended an early long heavy bone graft. In reply *Mr Cloke* mentioned that stability was not obtained with cancellous chips. He preferred to avoid a second incision over the tibia or iliac crest.

Complications of internal fixation of fractures of the tibia—*Mr H. Barry* (Sydney) had reviewed the tibial fractures treated by operation at one hospital during the years 1950–55. In this time 127 tibial fractures were treated in the operating theatre and of these fifty-one were plated (40 per cent). Bone grafts were performed in eighteen patients, of whom twelve had been treated initially by plating. A comparison with another large metropolitan hospital, where conservative treatment was much more commonly used, testified strongly that early metal fixation of fractures of the tibia predisposed to non-union. *Mr B. Keon-Cohen* (Melbourne), expressing inability to reduce spiral fractures of the tibia by closed methods, thought that the figures quoted could be misleading. *Mr F. Stonham* (Melbourne) thought the same. *Mr D. Sturrock* (Sydney) considered that non-union was no more unsatisfactory than the effects of over-long immobilisation. *Mr T. King* (Melbourne) thought the pendulum had swung too far in surgical treatment of fractures. *Mr J. Lahz* (Brisbane) recalled that external damage was an indication for operation. *Mr Barry*, replying, said that arterial damage was his foremost indication for operation. Spiral fractures should be fixed with screws.

Contracture of tensor fasciae latae in old poliomyelitis—*Mr R. Hodgkinson* (Sydney) showed pictures of children with this deformity. He recalled Souttar's, Yount's, Irwin's and Barr's concept of the condition and their operations. He described preventive measures in acute cases, and operative management in late cases: in children division of the iliotibial band, in adults or badly neglected children a division also of the tight structures in front of the hip. *Mr H. Crawford* (Brisbane) had seen Ober at work, and had had good results from Barr's operation. *Mr E. West* (Adelaide) said that the anterior superior iliac spine should be removed to facilitate closure of the wound. *Mr J. Colquhoun* (Melbourne) had become conservative in correcting hip flexion by operation. *Mr D. Parker*

(Hobart) recalled the method of Dame Agnes Hunt. *Mr A. R. Hamilton* (Sydney) recommended a Jones frame in preference to a plaster spica in the convalescent stage. *Mr Hodgkinson*, replying, pleaded for early recognition of the condition in children. Physiotherapy could not correct established deformity, and early simple operation was necessary.

Operations on painful feet—*Mr B. Keon-Cohen* (Melbourne) described the operative treatment of painful feet, including multiple excision of metatarsal heads in feet deformed by rheumatoid arthritis. *Mr F. Callow* (Sydney) advocated excision of the corn-bearing area in such feet. *Mr H. Crawford* (Brisbane) criticised lengthening of the extensor hallucis longus in Keller's operation. *Mr M. Gallagher* (Brisbane) asked Mr Keon-Cohen whether he had ever seen a permanent dropped toe. *Mr J. Kerridge* (Newcastle) recommended a small piece of oxycel gauze in the new joint space. *Mr L. Teece* (Sydney) emphasised that at least half of the proximal phalanx should be removed in Keller's operation. *Mr W. Hugh Smith* (Sydney) and *Mr A. R. Hamilton* (Sydney) discussed the advisability of Keller's operation in girls of twelve to fourteen years. *Mr N. Morgan* (Sydney) also used medullary wire fixation. He mentioned a case of slow infection in the wire track. *Mr E. West* (Adelaide) asked how he treated hallux rigidus in a working man. In reply *Mr Keon-Cohen* said he had no after-effects from division of the extensor tendon. He thought it important that a metatarsal bar be worn for three months after operation. He did not favour complicated tendon transplants. He did not do Keller's operation in young people. He occasionally removed the exostosis only.

Grice's operation for pes valgus—*Mr H. Barry* (Sydney) described cases of Grice's operation for subtalar fusion in valgoid feet. *Mr H. Crawford* (Brisbane) thought Grice's operation important. *Mr E. Price* (Melbourne) pointed out that Mr C. W. B. Littlejohn had been doing this operation for some years in Melbourne, and considered operation for postural deformities of the feet rarely necessary. *Mr E. West* (Adelaide) asked if the operation was done for spasmodic flat foot. *Mr H. Crawford* (Brisbane) recommended for this condition fusion of the first cuneiform and transfer of the tibialis anterior attachment, an operation with which he had had lasting success for twenty years.

Fracture-dislocation of the hip—*Mr H. Royle* (Sydney) showed a case of dislocation of the hip, which after reduction showed evidence of crushing of the head of the femur. *Mr F. Callow* (Sydney) said that this type of fracture often prevented closed reduction of the dislocation. *Mr J. Moloney* (Wollongong) mentioned a case that required open reduction because of this complication, in which myositis ossificans later developed. *Mr E. West* (Adelaide) agreed with the last speaker, and said that this type of fracture was becoming more common because of car accidents. *Mr W. Hugh Smith* (Sydney) congratulated the speaker on his conservative treatment. *Mr E. Price* (Melbourne) was in favour of conservative management if possible, but mentioned one case requiring open reduction in which the joint capsule was buttonholed. *Mr W. Goyton* (Melbourne) showed a dislocation of the head of the femur resisting reduction because of a fracture of the head, and also a case of central dislocation of the head of the femur reduced by heavy traction.

Congenital dislocation of the hip—*Mr A. McSweeney* (Brisbane) showed three cases of congenital dislocation of the hip in one family; there was no earlier family history. *Mr R. Wherrett* (Sydney), *Mr D. Parker* (Hobart), *Mr J. Colquhoun* (Melbourne) and *Mr W. MacDonald* (Sydney) described similar families. *Mr E. Price* (Melbourne), *Mr A. Hamilton* (Sydney) and *Mr M. Maxwell* (Sydney) discussed methods of reduction and the incidence of subsequent " Perthes " changes.

Cervical dislocation—*Mr E. Spring* (Melbourne) showed a case of spontaneous dislocation of the neck from a destructive lesion in the odontoid process. *Mr A. Hamilton* (Sydney) thought a bone graft would be necessary. *Mr J. Bryan* (Sydney) and *Mr E. Price* (Melbourne) described operative techniques in cervical fusion. *Mr Spring* showed also a case of traumatic atlanto-axial dislocation with later recurrence at football. Wire, used for fixation, broke, and a bone graft became necessary.

Scoliosis—*Mr M. Anderson* (Brisbane) showed a case of paralytic scoliosis treated by Risser jacket and Milwaukee brace and subsequent spinal fusion. *Mr E. Price* (Melbourne) described one fatal result. *Mr J. Cloke* (Melbourne) recalled that many of the patients presented " bad risks." *Mr F. Stonham* (Melbourne) mentioned the old method of attack through the abdomen for spinal fusion. *Mr D. Parker* (Hobart) favoured old-fashioned treatment of scoliosis with serial jackets.

Clinical cases. Congenital absence of the radius and thumb—*Mr D. Watson* (Brisbane) showed a case of this deformity. *Mr J. Lahz* (Brisbane) advised against operation. *Mr T. King* (Melbourne), *Mr E. Spring* (Melbourne), *Mr A. R. Hamilton* (Sydney), *Mr H. Barry* (Sydney) and *Mr E. Price* (Melbourne) discussed similar cases in their practice and their operative techniques in dealing with it.

Adamantinoma—*Mr J. Lahz* (Brisbane) showed a case of adamantinoma of the right mandible. *Mr E. Spring* (Melbourne), Mr *B. Keon-Cohen* (Melbourne) and *Mr F. Callow* (Sydney) described cases of adamantinoma in other sites, the bad prognosis and attempts at massive bone replacement with limited success.

Bilateral coxa valga, with slipped epiphysis—*Mr J. Lahz* (Brisbane) showed a case. *Mr B. Keon-Cohen* (Melbourne) and *Mr E. West* (Adelaide) had divided the femoral neck for coxa valga with deplorable results. *Mr A. R. Hamilton* (Sydney) recommended subtrochanteric osteotomy. *Mr T. King* (Melbourne) thought the displacement not severe enough to worry about but that the patient should avoid weight bearing till the epiphyses fused. *Mr R. Stephen* (Sydney) and *Mr N. Wilson* (Adelaide) quoted poor results from operations to replace the epiphysis.

Fractured neck of talus—*Mr R. Hodgkinson* (Sydney) showed a fracture of the neck of the talus. *Mr J. Colquhoun* (Melbourne) thought the head of the talus should be removed. *Mr W. MacDonald* (Sydney) and *Mr W. Hugh Smith* (Sydney) advised conservative treatment. *Mr M. Gallagher* (Brisbane) asked how long weight bearing should be avoided. *Mr T. King* (Melbourne) advised conservative treatment for the time being, though probably a compression arthrodesis of the ankle, subtalar joint and other tarsal joints (a quadruple arthrodesis) would be necessary later.

" Quadricepsplasty "—*Mr J. Anderson* (Brisbane) showed two cases in which quadricepsplasty was done for stiff knees after femoral fracture. *Mr B. Keon-Cohen* (Melbourne), *Mr D. Sturrock* (Sydney) and *Mr F. Callow* (Sydney) thought it a useful procedure.

" Carbuncle " of the hip—*Mr T. Stubbs-Brown* (Brisbane) described a case of carbuncle of the left hip with good response to chemotherapy. *Mr R. McKellar Hall* (Perth) advised continuance of conservative measures. *Mr D. Parker* (Hobart) recalled a similar case with pyaemia and osteomyelitis of the ilium involving the hip. *Mr F. Callow* (Sydney) recommended full movement now in this case but no weight bearing.

Leg equalisation—*Mr A. Crawford* (Brisbane) showed a case in which discrepancy due to a short leg was treated by shortening of the other leg (Phemister's graft) and ablation of the fibula, with later stapling of the femoral epiphysis; an inequality of two and a half inches had been fully corrected. *Mr E. West* (Adelaide) was unhappy about the results of stapling. *Mr E. Price* (Melbourne) agreed and now preferred the Phemister procedure. *Mr A. R. Hamilton* (Sydney) preferred leg shortening to lengthening.

Avascular necrosis of the capital femoral epiphysis—*Mr N. Wilson* (Adelaide) showed a case of Perthes' disease in a ten-year-old healthy boy. *Mr G. Colvin* (Sydney) thought that widening of the neck of the femur was diagnostic of this condition, but *Mr W. MacDonald* (Sydney) disagreed with the diagnosis and considered that the wandering acetabulum suggested an infective arthritis, and *Mr E. Price* (Melbourne) postulated avascular necrosis from a fall. *Mr A. R. Hamilton* (Sydney) mentioned an association of incontinence of urine with Perthes' disease, but *Mr J. Colquhoun* (Melbourne) thought there was no important association. *Mr R. Hodgkinson* (Sydney) asked if the patient had been given cortisone for the treatment of associated asthma.

Osteosarcoma—*Mr R. Hodgkinson* (Sydney) showed radiographs of an osteogenic sarcoma of the femur. *Mr A. Hamilton* (Sydney) suggested needle biopsy. *Mr B. Keon-Cohen* (Melbourne) thought this procedure dangerous and that an adequate biopsy was necessary. *Mr F. Callow* (Sydney) and *Mr H. Barry* (Sydney) stressed the bad prognosis, considered amputation of little value and preferred radiotherapy in most cases.

Fibrous dysplasia and cysts—*Mr J. Colquhoun* (Melbourne) showed radiographs of a simple cyst of the neck of the femur, and also of a seemingly multiloculated cyst of the upper end of the femur with short leg and scoliosis. *Mr A. R. Hamilton* (Sydney) showed radiographs of fibrous dysplasia of the hip with involvement of the acetabulum. *Mr R. Wherrett* (Sydney) showed a " cyst " of the upper end of the femur considered to be a benign ossifying fibroma, the contents at operation looking like a " primrose flummery." *Mr D. Parker* (Hobart) showed a malignant tumour of the upper end of the humerus thought to arise from malignant change in fibrocystic disease; biopsy was difficult and required ligation of the subclavian artery to control haemorrhage. *Mr W. MacDonald* (Sydney) described fibrous dysplasia in three children. In one, the involved lower third of the tibia presented bowing, which gradually disappeared spontaneously. *Mr A. R. Hamilton* (Sydney) described a patient with fibrous dysplasia treated by bone grafting from the father. *Mr M. Gallagher* (Brisbane) showed two cases of bone cyst, one in the ulna, with pathological fracture, and the other in the lower end of the fibula.

Sequelae of burns—*Mr W. MacDonald* (Sydney) showed a child in whom severe burns requiring extensive skin grafting had been followed by ossification about the hips with consequent ankylosis of the one more affected. *Mr J. Colquhoun* (Melbourne) showed a child who had lost both legs through the knees from burns.

Other cases shown included: examples of osteomyelitis by *Mr R. McKellar Hall* (Perth), *Mr L. Teece* (Sydney) and *Mr I. McPhee* (Brisbane); dislocation of the head of the radius, and a crushed arm with subsequent median nerve paresis relieved by carpal tunnel decompression, by *Mr G. Colvin* (Sydney); excision of middle third of the ulna and skin-grafting after a gunshot wound of the forearm,

by *Mr D. Parker* (Hobart); relief of sciatica after excision of an osteophyte projecting into the L.4–5 intervertebral foramen, by *Mr C. Hudson* (Sydney); undiagnosed tumour of the medial condyle of the femur in a patient with extensive pulmonary tuberculosis, by *Mr B. Keon-Cohen* (Melbourne); avulsion fracture of the calcaneum, by *Mr L. Teece* (Sydney); eosinophilic granuloma of the femoral shaft, by *Mr J. Colquhoun* (Melbourne); secondary neoplasia of the femur, by *Mr L. Teece* (Sydney); osteogenesis imperfecta with gross anterior bowing of the femur and 90 per cent correction by supracondylar wedge osteotomies, and examples of supracondylar osteotomies performed for knock-knee in Brisbane in 1889, shown by *Mr A. McSweeney* (Brisbane); arthrographs of knees and of the shoulder to demonstrate the defect in the humeral head in recurrent dislocation, by *Mr F. Stonham* (Melbourne); radiographic bone changes in a cretin, by *Mr A. R. Hamilton* (Sydney); osteochondromatosis of the elbow seen four months after injury, by *Mr W. Gayton* (Melbourne).

Gonadal protection against x-rays—*Mr R. Wherrett* (Sydney) demonstrated a shield for the genitals of a child who is likely to be subjected to much irradiation, for instance a child with congenital dislocation of the hip.

DENMARK

REVIEW OF ORTHOPAEDIC ACTIVITIES

Dr J. Mortens has sent us the following report of orthopaedic activities in Denmark.

Orthopaedic appointments—*Dr Hj. Larsen* has been appointed to succeed the late Sv. Kiær as senior surgeon at Department II, the Orthopaedic Hospital, Copenhagen. *Dr B. Eriksen* has been appointed his successor as junior surgeon at the Orthopaedic Hospital, Aarhus. *Dr K. Bang Rasmussen* has been appointed junior surgeon at Department I of the Orthopaedic Hospital, Copenhagen.

The lectureship in orthopaedic surgery at the University of Copenhagen, of which the previous holders were Paul Guildal and then *Aage Berntsen* and which is now held by *Dr A. Bertelsen*, has now been upgraded to a professorship in orthopaedic surgery. *Dr A. Bertelsen* has been appointed to hold this chair at the University of Copenhagen.

The Society and Home for Cripples in Denmark—A Children's School and Home "Geelsgaard" has been opened just outside Copenhagen, the old school and home in Copenhagen at Esplanaden thereby being given better facilities as Vocational School and Home. The rehabilitation centre at Hornbæk now includes a spinal injury centre and at the orthopaedic hospital, Copenhagen, the majority of reconstructive hand surgery has been taken over by *Dr K. Bang Rasmussen*.

The professional staffing at present at the orthopaedic hospitals, clinics and rehabilitation centres is as follows: The Orthopaedic Hospital Copenhagen: Dept. I—*A. Bertelsen, W. Anthonsen* and *K. Bang Rasmussen*. Dept. II—*E. Hj. Larsen* and *K. Jansen*. Dept. III—*A. Monberg* and *J. Mortens*. Physical Therapy Department—*J. Bang*. The Clinic for Cerebral Palsy—*S. Brandt* (Neuropaediatrician, Chief of the Clinic), *J. Mortens* (Orthopaedic surgeon), *J. Bang* and *B. Sury* (Doctors in Physical Medicine). The Orthopaedic Hospital, Aarhus—*E. Thomasen* and *B. Eriksen*. The Orthopaedic Clinic, Odense—*Aa. Randløv*. The Orthopaedic Clinic, Kolding—*I. Lou*. The Orthopaedic Clinic, Aalborg—*P. Lütken*. The Rehabilitation Centre, Hornbæk—*O. Remvig*. The Rehabilitation Centre, Hald, Viborg—*N. Bukh*. Social Medicine—*A. W. Mortensen*.

DANISH ORTHOPAEDIC ASSOCIATION

At the spring meeting, 1956, the professional organisation of Orthopaedic Surgeons in Denmark was separated out from the Association, *A. W. Mortensen* (Copenhagen) being appointed chairman with *K. Jansen* (Copenhagen) as deputy and *J. Mortens* (Copenhagen) as treasurer. In the Danish Orthopaedic Association *A. Bertelsen* (Copenhagen) and *J. Mortens* (Copenhagen) were followed by *P. Lütken* (Aalborg) as president and *K. Bang Rasmussen* (Copenhagen) as treasurer; *J. Mortens* (Copenhagen) was elected vice-president.

A meeting of the association was held in Copenhagen in October 1955.

Requirements for hip prosthesis in replacement arthroplasty—*R. Movin* (Copenhagen) put up the following requirements: 1) Immunity to tissue reaction. No wear must take place creating foreign body reaction. (Nylon in this respect is inferior to acrylic in spite of the fact that in experiments outside the body the wear on nylon was found less than that on acrylic.) 2) Resistance to oxidation. 3) The shape of the prosthesis is important if luxation, loosening of the prosthesis or insufficiency of the gluteus medius is to be avoided. The use of acrylic cement to fix the prosthesis has neither clinically nor by experiments proved to be harmful and is therefore still in use at the hospital.

Charnley's central-dislocation operation for osteoarthritis of the hip—*J. Mortens* (Copenhagen) reported twelve patients operated upon at the Orthopaedic Hospital, Copenhagen, and observed for six to

fourteen months. No internal fixation or compression was used. In two younger patients the aim had been to produce a bony fusion and a long hip plaster including the knee was maintained for two months before weight bearing was permitted in a short plaster. Fusion was established in the one and was probable in the other. In the other ten cases the patients were older—between fifty-six and sixty-eight years. They were allowed to put weight on the leg in a short spica after three or four weeks, the plaster being discarded six to eight weeks after operation. The aim had been to avoid post-operative stiffening of the knee in these old patients and to allow them up early. The object was not primarily to produce bony fusion but a fibrous ankylosis with a " bone block for adduction." It was felt that better stabilisation of the medial shift of the femur could be produced in this way than by the usual displacement osteotomy, and with less risk of knee stiffness and earlier ambulation. Four patients had excellent results, with a painless firm fibrous (in one probably osseous) ankylosis. All these were back at work three to five months after operation. Three patients were improved but experienced slight pain when starting walking and used a stick. Two patients developed a creeping fracture; one was nevertheless definitely improved, the other was not. One patient died from a pulmonary embolus. It was felt that these results warranted further use of the method in cases of osteoarthritis of the hip with a normal spine. In cases complicated with arthrosis of the spine a displacement osteotomy fixed with the Kessel spike-plate was performed.

Compression arthrodesis of the hip—*R. Movin* (Copenhagen) had performed a compression arthrodesis of the hip with an apparatus similar to Charnley's, but without creating a hold through the pelvis, in thirteen cases, of which seven were cases of failed acrylic arthroplasty. Sound fusion was obtained in nine cases, one had failed, and one had a painless fibrous ankylosis with some mobility. Two operations were done too recently to allow assessment of the result.

Buschenfeldt's corset in treatment of scoliosis—*J. Saugmann-Jensen* (Copenhagen) reported a series of cases of paralytic and idiopathic scoliosis in which the Buschenfeldt corset had been tried in the hope of preventing further deformity. No certain conclusions could be drawn.

Spasticity during sleep in children with cerebral palsy—*A. Jørgensen* (Hald, Viborg) reported a clinical study of the spasticity in cerebral palsied children. He found that the spasticity decreased in many children during sleep.

A meeting was held in Copehagen in May 1956.

Treatment of congenital club foot—*A. Bertelsen* (Copenhagen) had the impression from old and modern literature from all over the world that the results of manipulative treatment instituted early had not improved, the unsatisfactory results generally being about 25 per cent. Advocates of special methods, such as Denis Browne or Kite perhaps obtained somewhat better results in their own series. He reviewed 114 feet treated initially by manipulation after the method of Thomasen or Denis Browne in the years 1947–52. The results after half a year's treatment showed only thirty-four complete cures. Late results were good in 44 per cent, satisfactory in a further 28 per cent and bad in 28 per cent. To obtain these results soft-tissue operations were performed on thirty-nine of the 114 feet, only nine having been done within the first six months. He thought that the results might be improved by earlier soft-tissue operations, quoting Camera and Ré from Turin, who advocated operation from the age of six months in resistant cases.

K. Jansen (Copenhagen) reviewed 150 feet treated initially by Denis Browne's method in the years 1948–53. Sixty-five cases or 43 per cent showed good or satisfactory results, but supplementary measures were found necessary in the remaining cases, soft-tissue operations in sixty-one of these. The late results gave 50 per cent good, 38 per cent satisfactory and 12 per cent bad results. It was his opinion that regardless of which method of manipulative treatment was used a certain number of cases with inherent resistance would require operation.

W. Anthonsen (Copenhagen) gave a description of the method of Kite now in use at Department I, with only slight alterations from Kite's original method. It was too early to assess results.

Treatment of slipped femoral epiphysis—*K. Kristoffersen* (Aarhus) reviewed the patients treated at the orthopaedic hospital, Aarhus, in the years 1951–55. Ninety-six cases had slow progressive slipping and were treated by drilling the epiphysial plate. Fourteen patients had acute total slipping; open reduction was performed with wedge resection of the remoral neck, drilling of the head and fixation with a fine pin. In three of the fourteen cases avascular necrosis occurred. The anatomical results were good in eleven and fair in three cases. The functional results were good in eleven, fair in two and bad in one case. For comparison eleven cases from previous years with total slipping treated by closed reduction were reviewed. Three had avascular necrosis, five had a good or fair anatomical result, but no reduction had occurred in six cases. The functional result was good in eight, fair in two and bad in one case. It was felt that acute total slipping should be recognised as an entity and should be operated upon as mentioned above.

A meeting was held at the rehabilitation centre, Hornbæk, in November 1956.

Treatment and rehabilitation of patients with paraplegia from spinal cord injuries and diseases—*A. Bertelsen* (Copenhagen) reviewed the recent literature concerning the neurosurgical, plastic and orthopaedic problems, and *B. Eskesen* (Copenhagen) and *O. Remvig* (Hornbæk) demonstrated patients, methods of treatment and rehabilitation, and reviewed the results obtained at the Orthopaedic Hospital, Copenhagen, and at Hornbæk, in forty-three cases from 1950–54 observed up to October 1956. A comprehensive report is impossible but material and results might be summarised thus. Sixteen of the lesions were sequels of fractures of the spine, six were cases of transverse myelitis, ten of myelodysplasia, the rest cases of tuberculosis or tumours. Twenty-eight of the forty-three patients acquired a satisfactory bladder function. Pressure sores had occurred in thirty-three cases, often before admission, and sixteen needed plastic operations. Spasticity was reduced with certainty in sixteen. Bowel action was satisfactory in all cases but three out of four patients had incontinence of flatus. Eighteen were able to get about out of doors without help, fourteen could walk only small distances indoors but could manage their toilet without help. Eight could get about only in a wheel chair, and three were still bedridden. Two-thirds of the patients at present had no social problems, but many of them were children who could attend school. Dwelling problems were difficult.

GERMAN DEMOCRATIC REPUBLIC

ORTHOPAEDIC ASSOCIATION OF THE GERMAN DEMOCRATIC REPUBLIC, 1957 MEETING

The Seventh Congress of the Orthopaedic Association of the German Democratic Republic was held in Leipzig from May 9–11 under the presidency of Professor P. F. Matzen.

Eighty papers were given. The main subject discussed was congenital dislocation of the hip—its conservative and operative treatment and the treatment of its late results. Other subjects discussed in detail were developmental abnormalities, enchondral dysostoses, and the operative treatment of flat foot. In addition, papers were given on various aspects of such matters as scoliosis (causation and operative treatment), ankylosing spondylitis and arterial disease.

An exhibition of films on orthopaedic subjects was given on the evening of May 9. Operative techniques for the correction of scoliosis and for arthroplasty of the hip were shown, and other films dealt with such subjects as replacement of the amputated thumb and the fitting of limb prostheses.

During the Congress the University Clinic for Orthopaedic Surgery was open for inspection by visitors, and an exhibition of orthopaedic instruments and appliances was also available for study.

Among the enjoyable social activities were the informal evening party held at the opening of the Congress, and the banquet which took place on the evening of May 10 at Markleeberg.

Both members and guests of the orthopaedic Association attended the Congress and gave papers and took part in the discussions. Among the guests were surgeons from America, Austria, Bulgaria, Czechoslovakia, England, Finland, the German Federal Republic, Hungary, Italy, Poland, Russia, Spain and Switzerland.

ANNOUNCEMENTS

BRITISH ORTHOPAEDIC ASSOCIATION SCHOLARSHIP 1958

The British Orthopaedic Association invites applications for a Travelling Scholarship of approximately six months' duration to visit one or more centres in the United Kingdom or abroad (excluding the dollar area). The sum available will be not less than £500, and may be increased as necessary for living expenses and travel.

The object of the visit, the proposed itinerary, and the previous clinical experience and research contributions of the candidate, will be major considerations in making the award.

Applications should be made to the Secretary, British Orthopaedic Association, 45 Lincoln's Inn Fields, London, W.C. 2, not later than November 1, 1957. Short-listed candidates will be interviewed on December 6, 1957. Application forms may be obtained at the above address.

CASES OF SPECIAL INTEREST

The Editorial Board is prepared to allow space in the Journal for requests from individual surgeons who wish to collect reports of uncommon injuries or diseases in which they have a special interest. Any reader who has material relating to the subject mentioned below and who is willing to pass it on for the common interest is asked to communicate with the Editor, or directly with the surgeon concerned.

Simultaneous fractures of both femoral necks—Case reports or information relating to simultaneous fractures of both femoral necks, particularly in association with electro-convulsive therapy, is requested by *Mr H. D. W. Powell*, of 18 Birch Polygon, Rusholme, Manchester 14, who is studying the incidence of this injury.

Book Reviews

INJURIES OF THE HAND. By Ronald FURLONG, F.R.C.S., Assistant Orthopaedic Surgeon, St Thomas's Hospital; Orthopaedic Surgeon, Rowley Bristow Hospital, Pyrford, and Queen Victoria Hospital, East Grinstead. $10 \times 6\frac{3}{4}$ in. Pp. vii+215, with 99 figures. Index. 1957. London: J. & A. Churchill Ltd. Price 36s.

This is an excellent short text-book on hand injuries, with most of the emphasis on the problems of tendon surgery. As one has come to expect from the Orthopaedic Department of St Thomas's Hospital, the approach is straightforward and practical. There is no blind adherence to tradition, and the methods of treatment advocated are clearly the result of experience—they have been included because they work. Here and there the advice contained is dogmatic, and alternative methods are briefly discussed or perhaps not mentioned. Dogmatism, however, is surely one of the essentials of elementary teaching, and this book loses nothing by being positive in its approach. It is written in a pleasant, if racy, style, and is admirably illustrated; indeed the reviewer was most impressed by the quality and variety of the photographic illustrations and by the virtual absence of diagrams.

If there is to be any criticism it must, I feel, be on the subject of emphasis. In the publishers' blurb the book is described as being " for the junior surgeon who deals with hand injuries in Casualty Departments. It should also prove of inestimable value to general practitioners in districts where

(a) FIG. 86 (b)

These elastic garters are simple to make and are effective in use.

casualty surgery at cottage hospitals forms part of their duties "—perhaps an unfortunate reference, when one considers the emphasis the author himself rightly places on surgical skill and experience in the primary treatment of the injured hand. The description of the evaluation and treatment of major hand injuries is surely inadequate. The author explains this on the grounds that such subjects as the replacement of large areas of skin loss are the proper province of the plastic surgeon, but this argument does not apply where plastic surgical experience is not immediately available. Surely, too, it loses some of its force when the subject of tendon injuries is dealt with so thoroughly and in so great detail. The primary treatment of the major hand injury is just the problem that may confront the surgeon who is on his own. There is in the evaluation of the extent of injury, in the ingenuity

required to obtain skin cover, and in the estimate of final function, for which the primary operation is so vital a preliminary, all the art of surgery compressed into a small compass. In this book the problem is indeed tackled, and with proper emphasis on careful cleansing and excision, primary skin cover, and the pre-eminent importance of function in the plan for treatment; nevertheless, it would have been better had these subjects been dealt with in greater detail.

The problem of tendon injuries is most beautifully described and illustrated. The author's results in tendon surgery have long excited our admiration, and this book gives some indication of how these results have been achieved. One typical example of the practical nature of the instruction contained in it is the elastic garter splint for assisted active finger exercises—how much simpler than the elaborate rehabilitation methods which have been described, and which are beyond the scope of the average physiotherapy department (Fig. 86). The practical common-sense approach to the surgery of hand injuries is indeed the most striking feature of this book, and it is strongly recommended to all interested in the subject.—E. Mervyn Evans.

CHIRURGIE ORTHOPÉDIQUE DES PARALYSIES. By R. Merle D'Aubigné, Professeur de Clinique chirurgicale orthopédique et réparatrice à la Faculté de Médecine de Paris; J. Bénassy, Chirurgien-Assistant des Hôpitaux de Paris; and J.-O. Ramadier, Chirurgien-Assistant des Hôpitaux de Paris, with twelve other collaborators. 10×7 in. Pp. 431, with 171 figures. Index. 1956. Paris: Masson et Cie, Éditeurs. Price Broché 4,200 fr. Cartonné toile 5,000 fr.

The surgical relief of paralytic disorders—an important aspect of orthopaedics—has steadily advanced in recent years. Those who wish to master it must be rather more than amateur neurologists and possess soundness of judgment well above the average. The number of books on the surgery of paralysis is small, perhaps because it is so hard to tackle the subject systematically and also because few people have experience in the treatment of every variety of paralytic disorder. So this book by Professor Merle d'Aubigné and his colleagues is unusually valuable. Those who expect to find an encyclopaedia will be disappointed; for example, the repair of nerves is dismissed in twenty-two pages, the treatment of post-poliomyelitic paralysis of the forearm and hand is dealt with summarily; and there is nothing about the treatment of the more benign progressive disorders such as peroneal muscular atrophy. This is rather a collection of essays by many writers each dealing with a topic that he has mastered thoroughly; if this is appreciated the reader will be satisfied.

One of the happiest features of this book is the balanced account it gives of French, American and British practice, and the appreciation expressed by our Parisian colleagues of work done on the other side of the Channel and of the Atlantic is in places almost embarrassing in its warmth. This is yet another expression of the splendid relationship in the field of orthopaedics that has developed so rapidly since the war between those who speak French and those who speak English, and on the French side it has been inspired by one person pre-eminently—Merle d'Aubigné himself.

The book is divided into the following sections: paralysis of the upper limb; paralysis of the lower limb; painful amputation stumps and causalgia; paraplegia; hemiplegia in the adult; spastic paralysis in children; the treatment of poliomyelitis; and, lastly, a section on electrodiagnosis and physiotherapy in the management of peripheral nerve lesions.

The largest section is concerned with the upper limb and is outstandingly good, especially the chapters devoted to the palliative treatment of nerve injuries and to Volkmann's contracture. The reconstructive surgery of the lower limb is in itself a less lively subject, and here it is represented by only two sections, one on the palliative surgery of sciatic paralysis, the other on operations on the foot after poliomyelitis. The surgery of painful amputation stumps and of causalgia is discouraging and uncertain; the account of it given here is about as clear as anything on the subject can be. In the discussion on the interruption of central tracks for the relief of painful stumps the argument leading to the conclusion that antero-lateral cordotomy is the best operation is set out most elegantly.

Three conditions are covered in the section on paraplegia: injuries of the spinal cord, Pott's paraplegia and a postscript on paraplegia in scoliosis. Guttmann's excellent work is warmly commended but Holdsworth's break with strict conservatism in the early treatment of traumatic paraplegia is fairly appraised too. At the end this is what we are told: " Operation should not be attempted except under conditions where both nursing and equipment are ideal for treatment by internal fixation of the spine. No operation should be regarded in itself as a method for treating the paraplegia. If undertaken in general or special departments of surgery not organised for the care of paraplegics the most frightful things (they are listed) may and probably will happen." This is translated a little freely but it is what they mean, and it is plain common sense. Readers may, like myself, find matters of detail to quarrel about in this and other parts of the book, but they are as nothing in comparison

with the sanity of the authors' opinion and the notable absence of uncritical enthusiasm. The quotation given above is characteristic. Again, on Pott's paraplegia, although he goes very British, Merle d'Aubigné does not throw his hat in the air about antero-lateral decompression. Then Bénassy apologises for writing about the surgical treatment of hemiplegia (in otherwise fit young or middle-aged people) and goes on to present a perfect little essay on a much neglected subject. Although it might be argued that the chapter on the general treatment of poliomyelitis should find no place in this book, the fact that it is written by Grossiord is sufficient justification. Here is a real practical neurologist and the department he has built up at Garches is one of the best of its kind.

It is tempting to summarise many other parts of *Chirurgie Orthopédique des Paralysies* but perhaps it is better to leave the reader to look for himself. He will then discover a feature of this book that will endear its authors to the reader—their modesty. They almost carry it too far; we are not given even a glimpse of that great hive of activity at the Hôpital Cochin nor of the remarkable developments that have taken place during the last few years at Garches.

I cannot recall any British work on the treatment of paralysis since R. G. Gordon and Miss Forrester-Brown wrote their *Paralysis in Children* twenty-four years ago, an excellent book but covering rather a different field. So, until we stir ourselves, this book from France will be for us what Calot (not so modestly) called his text-book of orthopaedics—indispensable. And it is written with such lucidity that it can be read by anyone who has scraped through G.C.E. French—ordinary level.—H. J. SEDDON.

COMPENDIO DI MECCANICA ARTICOLARE (Summary of Joint Mechanics). By Irnerio FORNI and Osvaldo CAPPELLINI. 10 × 7 in. Pp. 354, with 250 figures. 1956. Bologna: Ed. Scientific Institute Rizzoli. Price 6,000 Lire.

The aim of this book seems more to diffuse acquired knowledge than to bring forward original thoughts or personal contributions. The work is in general well done, even if excessively confined to purely mechanical matters for only some passing reference is made to the biological influence of physical forces. No place is found in the book for the study of functional joint pathology, but this is not surprising. Even with these limitations the task that the authors set out to fulfil has been successfully concluded, namely to offer a suitable manual that may at a glance answer any question on the functional activity of any given joint and of the muscles responsible for its movements.—J. TRUETA.

STRESS AND STRAIN IN BONES. Their Relation to Fractures and Osteogenesis. By F. Gaynor EVANS, Ph.D., Associate Professor of Anatomy, Wayne State University College of Medicine, Detroit, Michigan. 9¼ × 6½ in. Pp. x + 245, with 42 figures and 19 tables. Index. Glossary of engineering terms. 1957. Springfield, Illinois: Charles C. Thomas, Publisher. Oxford: Blackwell Scientific Publications. Price 50s.

The author of this book is an authority on the subject to which he has repeatedly contributed during the last decade and his decision to write it must be gratefully acknowledged by all; since 1838, when F. C. Ward referred for the first time to the relationship between weight bearing and bone formation, to the present, the number of papers dedicated to studying the influence of pressure and tension on the shape and intimate structure of bone is incalculable, but good summaries are rare.

Dr Evans has selected well and written clearly on the subject. To the uninitiated this monograph will serve to clarify some frequently mistaken ideas, such as the supposition that stress is synonymous with force instead of the intermolecular resistance within an object to the action of an outside force applied to it, which is the description accepted by Dr Evans. Readers with some expert knowledge of the subject will enjoy this book, particularly the part describing the influence of stress and strain on moulding the embryonic bones and that concerned with the factors influencing the breaking strength of bones. Many readers may be surprised to know that the means by which the mechanical factors induce osteogenesis is still a mystery, as Dr Evans rightly points out, and it may well be that the reading of this monograph will induce new research on the organisation of the rigid structures of the animal body, a subject which even transcends the limits of biology to enter the metaphysical field. If this were to happen the author might feel handsomely repaid for his efforts.—J. TRUETA.

RHEUMATIC DISEASES, RHEUMATISM AND ARTHRITIS. By Heinrich G. BRUGSCH, M.D., F.A.C.P., Assistant Professor of Medicine, School of Medicine, Tufts University; Physician-in-Charge, Arthritis Clinic of the Boston Dispensary, a Unit of the New England Medical Center. $9\frac{1}{4} \times 6\frac{1}{2}$ in. Pp. xiv+330, with 58 figures and 24 tables. Index. 1957. Philadelphia and Montreal: J. B. Lippincott Company. London: Pitman Medical Publishing Co. Ltd. Price 80s.

This work provides an introduction to the study of rheumatic diseases from the medical point of view. As such it will be useful to students of orthopaedics, but such people will be disappointed if they seek for more than the most elementary statement of the principles and details of the orthopaedic management of diseases in which the orthopaedic surgeon must play an important part. Furthermore the book is strangely deficient in its discussion of those disorders of the locomotor system that are not strictly rheumatic, but which, as medical and surgical problems, require consideration in the differential diagnosis. It is, for example, very hazy upon tuberculosis of the joints. Extraordinarily little is said about manipulative treatment and other conservative procedures. The discussions of laboratory investigations, and of dietetic and medical care are excellent, but the radiographic illustrations are of poor quality.—Norman CAPENER.

MODERN TRENDS IN NEUROLOGY (Second Series). Edited by Denis WILLIAMS, *C.B.E.*, M.D., D.Sc., F.R.C.P., Neurologist, St George's Hospital, London; Physician, National Hospital, Queen Square, London; Civil Consultant to the Royal Air Force. $9\frac{1}{2} \times 6\frac{1}{2}$ in. Pp. xii+350, with 80 figures. Index. 1957. London: Butterworth & Co (Publishers) Ltd. Price 72s. 6d.

This is a successor to the series of essays published in 1951 under the title of *Modern Trends in Neurology*, reviewed in this Journal (vol. **33-B,** p. 650) in the same year. This second series contains twenty-four contributions, four of which are of direct concern to orthopaedic surgeons and all of which can be read with interest and profit even by those who are not neurologists. R. W. Gilliatt on Electromyography, F. J. Nattrass on Primary Diseases of Muscles and Hugh Garland on Diabetic Amyotrophy have produced papers which will be welcomed by their orthopaedic colleagues, but it is a pity that V. Logue uses only fifteen pages to discuss the problems of cervical spondylosis.

The book is well produced. A rather glaring mistake in the name of one of the authors should not have happened, particularly in what is a good collection of authoritative papers which one is glad to have.—D. Ll. GRIFFITHS.

SURGERY. A Guide to Surgical Diagnosis and Treatment Including Tropical Surgery. By W. G. KERR, M.B., F.R.C.S. (Ed.), Surgical Specialist, Tanganyika. $8\frac{3}{4} \times 5\frac{1}{2}$ in. Pp. viii+410. Index. 1957. London: Oxford University Press. Price 25s.

The penetration of western medicine into less advanced communities has led to a demand for medically qualified men and women that cannot be fully met until the younger Universities and University Colleges that are doing such good work in those parts can expand with the passage of time and the continued expenditure of effort and money. In the meantime, a considerable burden of medical work must fall upon auxiliaries, many of whom, in the British Commonwealth, enjoy a remarkably good training within a limited field, thanks to the labours largely of their medical brethren and nursing sisters. There has naturally arisen a demand for books on western medicine suited to their particular needs, and it is fortunate that the Oxford University Press has now undertaken to meet this need. The first book—on surgery—promises very well. It is remarkable what an amount of sound, practical teaching, as well as basic information, Mr W. G. Kerr has succeeded in introducing into this short guide. Naturally, much of it has to do with injuries and surgery of the limbs. These are well tackled within the space available. Here is a worthy aid to those who are doing pioneer work in the less endowed parts of the world, especially the tropics. The book is in most commendably simple English, and perhaps this is some excuse for the omission of illustrations. As a small matter of detail it is perhaps worth mentioning that, although the Thomas's knee splint is given its full place, no mention is made of the screw distraction splint for the neck, which might well be called the Thomas's neck splint as it is the key to moving safely the patient with a broken neck. In the open spaces the safe transport of the patient under difficult conditions is often the key to successful treatment.—H. Jackson BURROWS.

POSITIONING IN RADIOGRAPHY. By K. C. CLARK, *M.B.E.*, F.S.R., Principal of the Department of Radiography and Medical Photography of Ilford Limited. Seventh edition. 12×9 in. Pp. 655, with 2,150 figures. Index. 1956. London: Ilford Limited. William Heinemann (Medical Books) Ltd. Price £5, 5s.

This handsome volume is likely to be found as a standard work of reference in most departments of radiodiagnosis. This new edition of a work already widely accepted as being of outstanding value comes appropriately to encompass the considerable advances in technique which have been made since the sixth edition appeared in 1951. It is noticeably larger than its predecessor, having expanded by 128 pages and no less than 682 illustrations. Its solid binding ensures that it will stand up to constant use.

The book covers the whole field of radiography, but those working in orthopaedic radiology departments can be grateful to Miss Clark for her detailed treatment of their special subject. The section on the cervical spine, for example, has been expanded and is entirely adequate. Shortcomings, however, can be found in the discussions on arthrography, where some illustrations would seem to be desirable and where the radiographer might be warned of the frequent adverse reactions in adults during such examinations of the shoulder. Some radiologists may disagree with the technique described for myelography.

As the work is stated to be concerned entirely with technique, and neither apparatus nor diagnosis, it is reasonable that such matters as high kilovoltage technique and high ratio grids should receive

FIG. 519

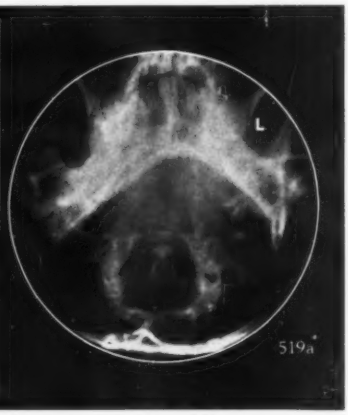


FIG. 519a

only passing references. But, though Miss Clark rightly considers that diagnosis is not the main concern of the radiographer, the reviewer has found that the good radiographer is indeed interested, and it seems a pity that only some of the excellent illustrations, which incidentally show pathological conditions, carry an accompanying diagnosis.

But these are minor criticisms. Practically all the procedures employed in orthopaedic radiography are well and adequately described. Reference is made to modern methods of true limb length measurement and to the examination of the spine in scoliosis. A new section is included on macroradiography, with an open assessment of its practical value. Enough is said of the new image intensifier and cineradiography to remind us that, in this equipment, we have a new tool for the examination of joint function and this may well prove a feature in the next edition. The increasing use of tomography for skeletal examination also attracts attention; and, in conjunction with this, it is wise to consider the word of warning about radiation exposure. This includes a nomogram for the calculation of radiation dosage, and it would be well for readers themselves to use this in order to appreciate the amount of radiation to which the patient is subjected in, for example, full examination with tomography of the lumbo-sacral region. While this dosage can be reduced by the use of suitable filters, high kilovoltage technique and, at a small sacrifice of detail, fast films and intensifying screens, the greatest single advance promises to be the use of multisection tomography. With this auxiliary apparatus the amount of radiation employed for two single ordinary tomograms will produce a

simultaneous series of five layer images. This is now true of most sections of the skeleton, but further elaboration will be required to make it satisfactory for the examination of the lumbo-sacral region in a heavy adult.

This book is almost essential for every department of diagnostic radiology.—R. O. MURRAY.

MEDICAL NEGLIGENCE. Being the Law of Negligence in Relation to the Medical Profession and Hospitals. By The Right Hon. Lord NATHAN, *P.C.*, A solicitor of the Supreme Court; Chairman of the Board of Governors of Westminster Hospital; with the collaboration of Anthony R. BARROWCLOUGH, B.A., of the Inner Temple, Barrister-at-Law. $8\frac{3}{4} \times 6$ in. Pp. xxxii+218. Index. 1957. London: Butterworth & Co. (Publishers) Ltd. Price 35s.

It is unquestionable that medical men feel more insecure than ever before in relation to their legal liabilities to their patients. Nevertheless, the medical man, disquieted by the spate of actions against hospitals and doctors in recent years, may take comfort from the fact that if he possesses "reasonable" skill and uses it with "reasonable" care, he will be immune from damages for negligence, although indeed he may not be immune from suit by a disgruntled patient. This concept of reasonableness is fundamental to our common law. "The standard of care which the law requires is not insurance against accidental slips. It is such a degree of care as a normally skilful member of the profession may reasonably be expected to exercise in the actual circumstances of the case in question. It is not every slip or mistake which imports negligence." (per SCOTT, L. J., in *Mahon v. Osborne* [1939].)

Lord Nathan in this excellent little work has collected together all the important decisions that bear on medical negligence and has sought to extract from them the principles that now govern the common law on this subject. The orthopaedic surgeon will there learn that he may be held blameless, if, after carrying out some radical operation for malignant disease on the strength of a frozen section, he subsequently finds that the patient was suffering from some innocent condition; if, having delegated some routine duty in after-treatment to a competent junior, that junior carries out his duty negligently; or if he fails to have carried out a pre-operative blood count and the patient dies of haemorrhage because of an unsuspected leukaemia. On the other hand, he may be held liable if he fails properly to instruct his deputy as to after-treatment when he goes on holiday; if he fails to warn the patient of any serious operative risk; or if he experiments on his patient with some novel treatment. He will find, too, that it is his duty to keep himself reasonably abreast of developments but he is not necessarily negligent if he fails to read a single article.

If the surgeon's appetite has been whetted by these examples he should buy this book and he will be well rewarded in reading it, written as it is in simple and non-technical language and amply illustrated by "tabloids" of leading cases.

In a book of such all-round excellence it would be churlish to make too much of a statement in the preface that is not entirely accurate. Lord Nathan says, "It is an odd phenomenon of social psychology that, as long as the hospitals were still voluntary and the medical staff honorary, an action brought against a hospital or a medical man was something quite out of the way, yet since the National Health Service Act became operative in July 1948 there has been a remarkable and in some ways alarming flood of cases; and that notwithstanding that the hospitals are the same hospitals as before, and the medical men are, so to speak, the same medical men." In fact, the reason for the rarity of actions against hospitals and their staff before the 1940s was that, since *Perionowsky v. Freeman* in 1866, it seemed hopeless to bring an action in negligence against a member of the medical staff of a voluntary hospital who gave his services voluntarily and, since *Hillyer v. St Bartholomew's Hospital* in 1908, there was little chance that the patient who sued a hospital in respect of the professional negligence of its doctors or nurses would succeed. It was not until 1942, in *Gold v. the Essex County Council*, that this state of affairs was remedied, and even in that case only on appeal, because the Judge in the court of first instance was bound by *Hillyer's case*. It was thus after 1942, long before the Health Service started, that the cases against hospitals and their medical staff really began; and indeed the Minister was obliged to take over with the advent of the Health Service in 1948 a number of pending claims against hospitals and staffs consequent upon the decision in *Gold's case*. The fact is that the public conscience had been outraged at the position of almost immunity which had been built up in favour of hospitals and their staff so that a patient with a just claim seemed to have no chance of securing justice. That position has now been remedied and for a time the pendulum seemed to have swung in the opposite direction against the hospitals and their staff. However, there are signs that a balance has now been reached and that the flood of cases against hospitals is now abating.

This minor criticism in no way detracts from the value of this admirable book which should find a place on the bookshelf of every medical man on a hospital staff and every hospital administrator.—W. M. LEVITT.

DIE GESUNDE UND DIE KRANKE WIRBELSÄULE IN RÖNTGENBILD UND KLINIK. Pathologisch–Anatomische Untersuchungen. By the late Georg SCHMORL and Herbert JUNGHANNS. Fourth edition. 11×8 in. Pp. xii+330, with 419 figures. Index. 1957. Stuttgart: Georg Thieme Verlag. Price DM 86.

It is now twenty-five years since Schmorl died in 1932, the year of the first edition of this book. His work was already well known, but he was not destined to hear the world-wide acclaim which greeted this classic monograph. His object was to place the interpretation of radiological appearances in the spine on a firm basis of anatomy and pathology. As a worker in the latter fields he was not himself directly involved in the clinical aspect, which therefore received relatively minor attention. Schmorl's groundwork was prodigious; he had subjected some 10,000 spines to searching and detailed examination. He interested himself not merely in pathological conditions but also took the opportunity of studying the normal development of the spine; radiological appearances that had been considered of pathological significance were in many instances shown by him to be no more than variations within the normal stages of growth. He was the first to make a major study of the intervertebral

FIG. 237
Enlarged photograph of the sagittal section through a lumbar disc. Schmorl's nodes above and below and horizontal tear anteriorly.

disc; although he observed in many spines posterior herniations of the nucleus pulposus, their clinical significance escaped him and he concentrated his attention on those changes in disc and vertebra which lead to the formation of "Schmorl's nodes" or to the condition misleadingly called osteochondritis. Nevertheless he laid the foundations on which so much work on disc syndromes has been done in recent years.

The new edition contains some minor additions and alterations to bring it up to date, and some figures have been altered, but basically it is the same as the 1951 and 1953 editions. Professor Junghanns has felt free to lay greater stress on the clinical and functional aspects, but is to be congratulated on maintaining the original character of the work, a proper monument to Schmorl's industry. Forty-three closely printed pages of bibliography give some idea of the vast literature that has accumulated on this and allied subjects. The illustrations are superb; for these alone even those who read little German should peruse this book.—T. J. FAIRBANK.

DIE DIAFIXATION. By K. PAP, Director of the Orthopaedic and Accident Department of the University of Debrecen, Hungary. 9½×7 in. Pp. 252, with 124 figures. Index. 1956. Budapest: Hungarian Academy Press. Price 43s.

It is a pleasure to welcome such a well produced book from Budapest. Evidence of clear thinking, technical efficiency and a wide acquaintance with orthopaedic literature, both Continental and Anglo-American, is manifest. The title cannot be translated, being a compound word formed from "diaphysis" and "fixation." No claim is made to the development of a new idea, and the author is the first to avoid making any claim to priority.

The reduction and fixation of fractures of the long bones is recommended by the following method. Reduction is achieved by skeletal traction. Stability and close approximation of the fracture surfaces are achieved by the application of a modified Thomas's wrench, rotated so as to force the fracture surfaces together. Through an incision in the soft tissues and with the assistance of a hand-held pistol guide and a hand drill, a thick Kirschner wire is introduced at right angles to the fracture surfaces to transfix the fracture. Firm fixation of the fracture is claimed by this method, though an external plaster incorporating the wire is necessary in most cases. The method is thus a technical elaboration of methods used before—screwed pins, beaded wires or double pins. The physical characteristics of the fracture and the forces employed are analysed in detail and clearly illustrated. The chief disadvantage of the method is the need for constant radiographic screening in the theatre. The risks of exposure to the hands has deterred most surgeons from the regular use of screening methods and to this is now added the possible development of leukaemia. The method is not applicable in comminuted fractures. The disadvantage of crossed wires which tend to separate the two bone ends and thus produce non-union is emphasised. Convincing evidence of ability to achieve and retain suitable fractures in anatomical positions is given by a series of case reports, 416 treated cases being analysed. Most surgeons will, however, prefer the use of skeletal traction without transfixation of the fracture when there is only slight displacement, and the open introduction of fixation in the more difficult cases.

The book is beautifully produced and illustrated. A series of line drawings make every manoeuvre clear. The style is that of any standard German text-book and has been made familiar to most orthopaedic surgeons by the works of Böhler. For anyone interested in the mechanical aspects of fracture fixation, this book contains much useful advice; even though its precepts may not be fully accepted.—J. G. BONNIN.

THE TREATMENT OF FRACTURES. By Lorenz BÖHLER, M.D., Director of the Accident Hospital, Vienna; Professor of Accident Surgery, University of Vienna. Volume II. Translated from the thirteenth German edition by Otto RUSSE, M.D., and R. G. B. BJORNSON, M.D. $9\frac{1}{2} \times 7$ ins. Pp. viii+436, with 941 figures. Index. 1957. New York and London: Grune & Stratton. Price £6, 3s.

In reviewing the first volume of this new edition of Böhler's " Fractures " (this Journal, vol. **39-B**, page 176) I fell into error. I assumed that the second and third volumes would each equal the size of the first. I was wrong. The second volume has only 431 pages of text and five pages of index. At this rate the whole work may well be complete in 2,000 pages, only ten and a half times the size of the first edition. I anticipated a sixteen-fold increase; I spoke too soon.

Into these 431 pages the author has condensed, if " condensed " be the word, all that he has to say and most of what he has ever said about fractures of the femur and injuries of the hip. True, he saves a good deal of space by devoting only nine pages to the Küntscher nail in femoral fractures, the reader being referred to Böhler's earlier book on medullary nailing for the details of this technique. He makes no other concessions; certainly none to modernity. No well-tried methods are discarded. Any surviving devotees of the Whitman plaster will find here a seventeen-page account of its use, complete to the finest detail, including a list of 31 Questions We Should Ask Ourselves to Avoid Failures with this Method. These start with " Have I ordered all the (listed) material . . ., and have I checked it for completeness and quality? " Dislocated hips are still reduced under ethyl chloride anaesthesia, and a " thick rubber drainage tube " is put into infected hip joints after gunshot wounds.

Principles are vigorously emphasised. In case anyone missed them in Volume I, there are three more heavy-type announcements that the surgeon must aim at producing a little shortening in the treatment of shaft fractures.

The lists of mistakes to be avoided which were familiar to readers of earlier English editions are replaced by 23 lists of Questions We Should Ask Ourselves. Before contemplating the treatment of central dislocation of the hip we are reminded to Ask Ourselves " the same eight questions " as with any other type of dislocation, and four pages later we stop to Ask Ourselves another 23 specifically about central dislocation. They include " Have I avoided attempting manipulative reduction of pelvic fragments from within the rectum, as with Hegar's dilators? " After inserting a Smith-Petersen nail We Should Ask Ourselves 96 questions. The 96th is " Have I omitted removing the femoral head if reduction was possible with continuous traction? "

The account of fracture-dislocation of the hip is very good, and no one following this book could go wrong in the treatment of femoral shaft fractures by continuous traction. When one gets at the material one wants, it is usually excellent. One has to quarry rather a lot of rock to get at the diamonds, but it could have been worse. We might have had to Ask Ourselves if we had put the Smith-Petersen nail into the wrong patient.—D. Ll. GRIFFITHS.

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